"COMPARATIVE STUDY OF LOADING DOSE OF ANTIBIOTICS DURING INDUCTION OF ANAESTHESIA AND EMPIRICAL ANTIBIOTICS IN ELECTIVE CLEAN AND CLEAN CONTAMINATED CASES."

Dissertation submitted to

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In partial fulfillment of the

Regulations of the award of degree of

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Department of General Surgery

Coimbatore Medical College Hospital

Coimbatore – 641 018

CERTIFICATE

This is to certify that this dissertation titled **"Comparative Study of Loading Dose of Antibiotics During Induction of Anaesthesia and Empirical Antibiotics in Elective Clean and Clean Contaminated Cases."** submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch – I (General Surgery) is a bonafide work done by **Dr.R.Ranganathan,** post graduate student in General Surgery under my direct supervision and guidance during the period of September 2013 to August 2014.

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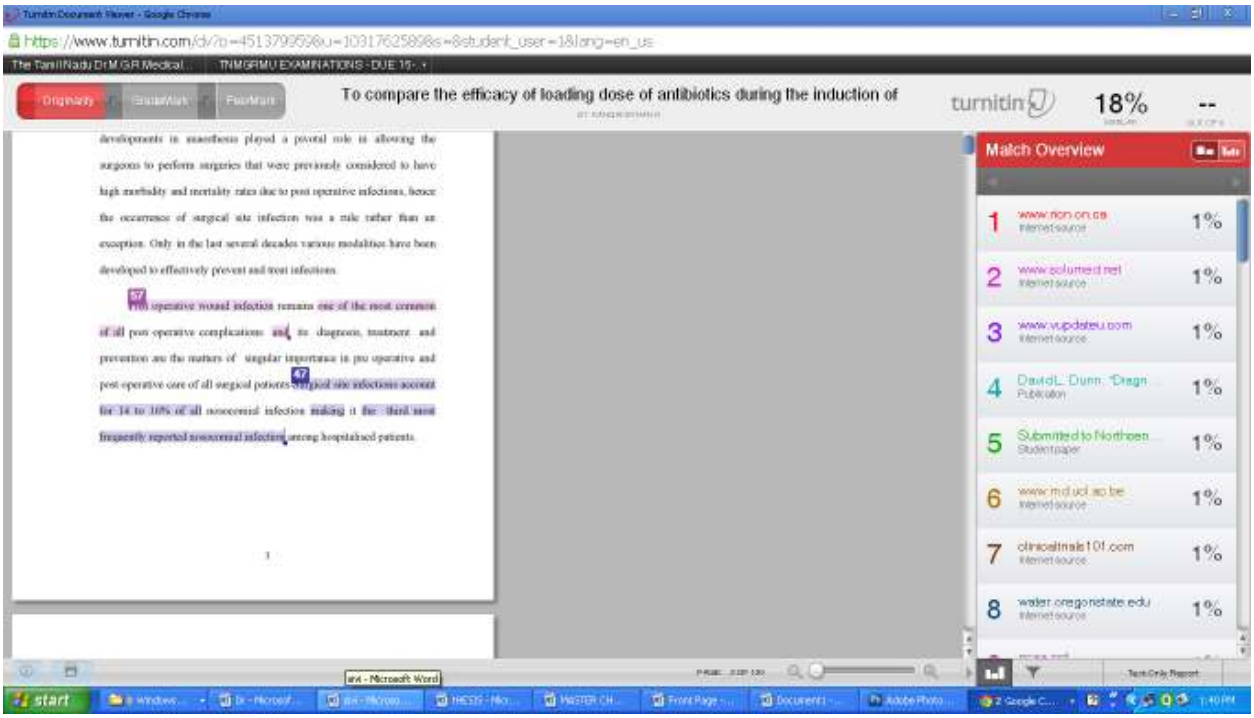
INTRODUCTION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developments in anaesthesia played a pivotal role in allowing the surgeons to perform surgeries that were previously considered to have high morbidity and mortality rates due to post operative infections, hence the occurrence of surgical site infection was a rule rather than an exception. Only in the last several decades various modalities have been developed to effectively prevent and treat infections.
Post operative wound infection remains one of the most common of all post operative complications and its diagnosis, treatment and prevention are the matters of singular importance in pre operative and post operative care of all surgical patients. Surgical site infections account for 14 to 16% of all nosocomial infection making it the third most frequently reported nosocomial infection among hospitalised patients.
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DECLARATION

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This dissertation is submitted to the Tamil Nadu Dr. M. G. R. Medical University towards the partial fulfillment of the University regulations for the award of M.S. Degree in General Surgery (Branch I).

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CONTENTS

INTRODUCTION

Since the dawn of time, the evolution of germ theory and antiseptics has led into the body of knowledge on the current field of surgical infectious diseases which has now become a vital component of the surgeon's practice. The germ theory and antiseptics along with the developments in anaesthesia played a pivotal role in allowing the surgeons to perform surgeries that were previously considered to have high morbidity and mortality rates due to post operative infections, hence the occurrence of surgical site infection was a rule rather than an exception. Only in the last several decades various modalities have been developed to effectively prevent and treat infections.

Post operative wound infection remains one of the most common of all post operative complications and its diagnosis, treatment and prevention are the matters of singular importance in pre operative and post operative care of all surgical patients Surgical site infections account for 14 to 16% of all nosocomial infection making it the third most frequently reported nosocomial infection among hospitalised patients.

Joseph Lister in the 1860's, established the rules of antiseptics following which post operative infection related morbidity substantially reduced. As a result of Listers work surgeries drastically reduced the sufferings and led to the prolongation of life.

The study of Holmes Pasteur and Koch in an infectious as well as in the operating room environment and the discipline described by Halsted continued to prove aseptic and antiseptic to be one of the first effective measures in prevention of infections in surgical patients.

The most common nosocomial infection among surgical patients was SSIs which accounted 38% of the total number of infections. Two thirds of the SSI restricted to site of incision and 1/3rd spread to involve spaces or organs approached during surgery. The observational studies on SSIs brought about awareness of the need of having knowledge on the appropriate use of aseptic and antiseptic techniques, with the proper use of therapeutic and prophylactic antibiotics and adequate observation and support with novel surgical, pharmacological as well as non pharmacological, aids to the present day modern surgeon. The prophylactic antibiotic therapy is evidently more effective when started preoperatively and continued throughout the intra operative period, with

the aim of maintaining therapeutic blood levels throughout the operative period. A single dose depending on the drug used and length of the procedure is often sufficient prophylactic antibiotic coverage for more than 12 hrs for a planned operation is never indicated.

AIMS AND OBJECTIVE

To compare the efficacy of loading dose of antibiotics during the induction of anaesthesia with empirical antibiotic usage in clean and clean contaminated elective general surgical procedures.

REVIEW OF LITERATURE

HISTORICAL REVIEW

The microbes are as old as the mankind itself. Nobody knows their origin on earth through out the history of mankind, treating infections has been one of the primary roles of a surgeon. Early in the history of mankind, there was recognition of inter play between wounds, infections and surgical manipulation. In fact, virtually all wounds became infected and infection was associated with high mortality.

Sushrutha, “Father of Indian Surgery” has also emphasized on the prevention of wound infection in his ancient scripts. The Edwin Smith surgical papyrus (1550 BC) and writings from Mesopotamia (1700-600 BC) make references to injuries, wounds and infections, and their treatment. The oldest known medical illustration is found in a tomb of a high ranking Royal of Sakkara in Egypt (2424-2262 BC). This door shows a physician (or priest) who is thought to be draining an abscess in the neck.

Our present understanding on surgical site infections has been contributed by the observations made by the nineteenth-century physicians and investigators. There have been two phases of intense revolutionary development in the means employed by surgeons against

infections[2]. The first of these two phases was centered on discovery of causes of infections and methods of its prevention. The great names associated with this phase are those of the fathers of bacteriology such as Pasteur, Robert Koch, and Joseph Lister. Second phase, was that of effective systemic treatment of the same. This phase is associated great names of Domagk and Florey.

From 1878 until 1880, Robert Koch explained the following four postulates to detect the association of organisms with specific diseases[3].

1. The suspected pathogenic organism should be present in all cases of the disease and absent from healthy animals.
2. The suspected pathogen should be isolated from a diseased host and grown in a pure culture in vitro Cells from a pure culture of the suspected organism should cause disease in a healthy animal.
3. The organism should be re-isolated from the newly diseased animal and shown to be the same as the original.

The scientific basis for the use of prophylactic use of antibiotics in surgery was laid by Miles and Burke in the late 1950s. They demonstrated that infections could be prevented only when AMAs (anti-microbial agents) were given prior to or at the time of infectious

challenge. They also concluded that AMAs administered after three hours after the infectious challenge were ineffective in preventing infection. Strachan and his colleagues performed the first prospective controlled trial, which investigated the proper post operative duration of administration of AMA in 1977. They concluded that there was no advantage of administering more than a single dose of AMA, preoperatively and no further doses were necessary postoperatively.

PATHOLOGY AND PATHOGENESIS

OF SURGICAL SITE INFECTIONS

The identification of SSI involves interpretation of clinical and laboratory findings, and it is crucial that a surveillance program use definitions that are consistent and standardized; otherwise, inaccurate SSI rates will be computed and reported. The Center for Disease Control's (CDC), National Nosocomial Infection Survey (NNIS) system has developed standardized surveillance criteria for defining SSIs. According to this criteria, SSIs were classified as incisional or organ/space. Incisional SSIs were further classified into Superficial incisional SSI, those involving skin and subcutaneous tissue and Deep incisional SSI, those involving deeper soft tissues of the incision.

CDC's CRITERIA OF SURGICAL SITE INFECTION (SSI)[4]

Superficial Incisional SSI

Infection involves skin or subcutaneous tissue of the incision site and occurs within 30 days after the surgery with one of the following present:

1. Associated drainage of pus, with the evidence of laboratory findings or in its absence, from the superficial site of incision.
2. Pathogens can be isolated from an aseptic sample of fluid or tissue obtained for culture from the superficial incision.
3. Presence of at least one of the following symptoms or signs of infection: pain, tenderness, redness, warmth or localized swelling.
4. Diagnosis of superficial incisional SSI by the surgeon or attending physician.

Deep Incisional SSI

Infection involves deep soft tissues, fascial and muscle layers, of the incision. Infection develops within 30 days after the surgery with no implant left at site or within 1 year with the implant in situ. The infection is related to the surgery with at least one of the following:

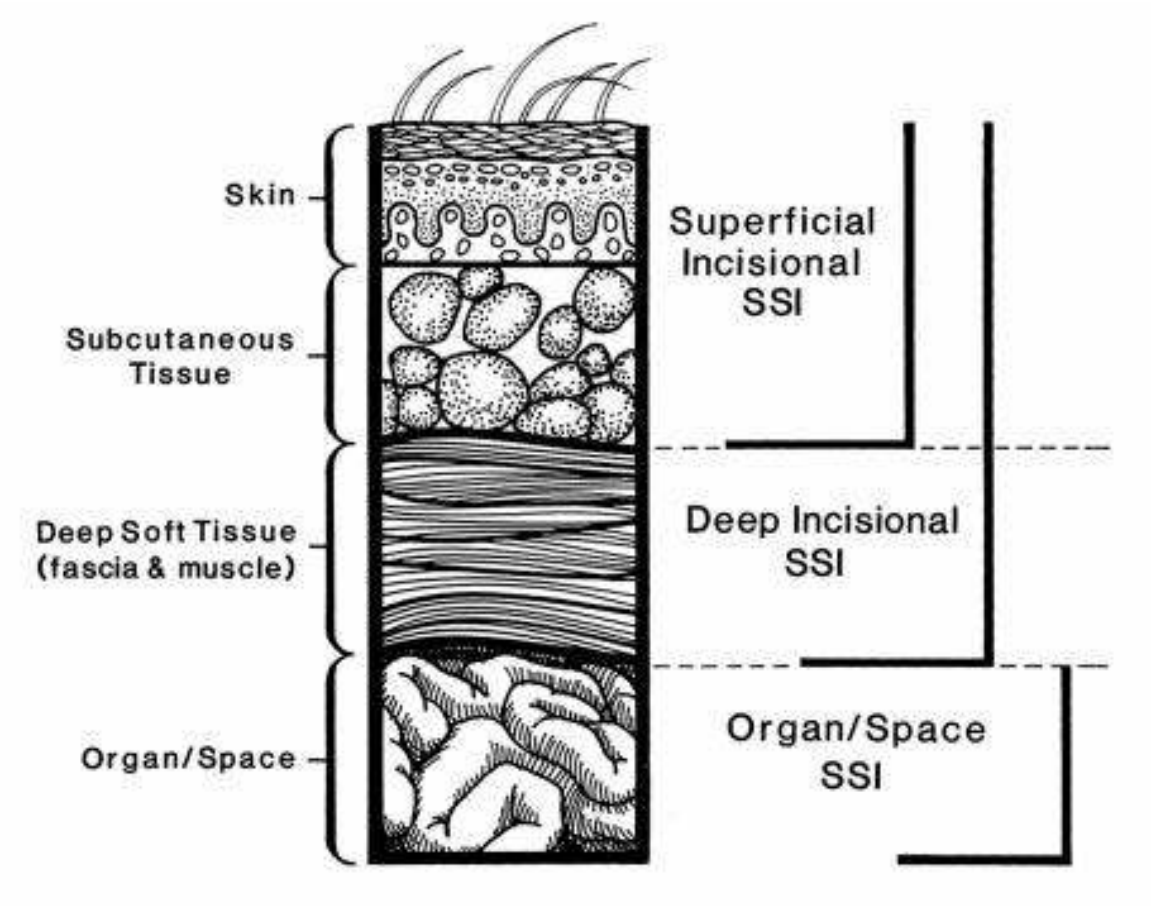
1. Associated purulent drainage from the deep incision but not from the organ/space element of the surgical site.
2. A deep incision is intentionally opened by a surgeon or spontaneously dehisces with the patient having at least one of the following signs or symptoms: Fever (>38ºC), localized pain or tenderness, except in case of incision site-culture-negative.
3. Deep incision having abscess or other signs of infection which is found on direct examination, during reoperation, or by histopathologic or radiologic examination.
4. Diagnosis of a deep incisional SSI by a surgeon or attending physician.

Organ/Space SSI

Infection involves any anatomical site, organs or spaces, except the site of incision, which was opened or manipulated during the surgery and develops within 30 days after the surgery with no implant left at site or within 1 year with the implant in situ. The infection is related to the surgery with at least one of the following.

1. Associated purulent drainage from a drain that is kept into the organ/space through a stab wound.
2. Pathogens can be isolated from an aseptic sample of fluid or tissue obtained for culture from the organ/space.
3. Organ/space having abscess or other signs of infection which is found on direct examination, during reoperation, or by histopathologic or radiologic examination.
4. Diagnosis of an organ/space SSI by a surgeon or attending physician.

CDC classification



SURGICAL WOUND CLASSIFICATION

Class I/Clean: An uninfected operative wound in which no inflammation is encountered and the respiratory, alimentary, genital, or uninfected urinary tract is not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Operative incisional wounds that follow non-penetrating (blunt) trauma should be included in this category if they meet the criteria.

Class II/Clean-Contaminated: An operative wound in which the respiratory, alimentary, genital or urinary tracts are entered under controlled conditions and without unusual contamination. Specifically, operations involving the biliary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in technique is encountered.

Class III/Contaminated: Open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (e.g., open cardiac massage) or gross spillage from the gastrointestinal tract, and incisions in which acute, non-purulent inflammation is encountered are included in this category.

Class IV/Dirty-Infected: Old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation

Determinants of Infection

Four determinants play an important role which lead to either complete healing of wound or SSI:

1. Bacterial Inoculation
2. Bacterial Virulence
3. Micro-environmental effects
4. Innate and acquired defences of host

Inoculum of Bacteria

Bacterial inoculation into the surgical site during the operation, is the determinant that has received the greatest amount of concentration. Pathogens may enter the wound from the atmosphere in the operating room, or by surgeons that come into contact with the wound or from the instruments used. Despite the thorough preparation of the skin bacteria are always present. Sites that normally are extensively colonized by bacteria, such as the bowel, are the largest source of contamination by

bacteria at the surgical site during the surgery. The distal small intestine and the colon have very large concentrations of bacteria with:

- 10^3–10^4 bacteria/mL of distal small bowel content
- 10^5 – 10^6 bacteria/mL in the right colon
- 10^{10} – 10^{12} bacteria/g of stool in the recto sigmoid colon

Old patients who have hypo- / achlorhydria, considerable numbers of bacteria are also present. In patients above 70 years of age or in patients having acute cholecystitis, or obstructive jaundice, or common bile duct stones. In surgeries involving female genital tract, concentration of 10^6 – 10^7 bacterial/mL is encountered.

Surgeries involving the oropharynx, lung, or urinary tract have significant bacterial contamination depending on the duration and type of disease that is responsible for the surgery. Particularly, SSIs generally occur as a consequence of intra-operative contamination and rarely occur as a result of bacterial contamination from distant infectious site through blood-borne seeding of the wound site during the postoperative period.

Virulence of the Bacterial Contaminant

Another factor leading to SSI is the bacterial virulence. The probability of infection is greater with more virulent bacterial contaminant. Coagulase-positive species, especially, Staphylococcal

species, need a smaller dose of infection compared to the coagulase-negative species. Clostridium perfringens and Streptococcus Group A need a smaller inoculum at the surgical site to cause severe necrotizing lesions. The virulence of Escherichia coli, the endotoxin, lies in its outer cell membrane. Bacteroides fragilis and other Bacteroides species ordinarily are pathogens of negligible virulence as solitary organisms, but along with other pathogens consuming oxygen, microbial synergism results and can cause considerable infection after procedures of colon or female genital tract.

The Microenvironment of the Wound

Another factor that is responsible for infection is the microenvironment of the wound. Products or consequences of the surgical procedure contribute to clinical infection by otherwise considered sub-infectious contamination of bacteria. A well known adjuvant substance is Hemoglobin, at the surgical site. A general consideration is that the liberation of ferric iron at the time of red blood cell degradation promotes proliferation of bacteria. Foreign bodies, particularly braided silk and other permanent braided suture materials, may also contain microbes and hence increase the likelihood of infection. Another adjuvant factor is dead space within the site of surgery which also creates a micro-environment that promotes infection.

The integrity of Host Defenses

Integrity of host defence system is another factor determining SSI. Weakened host defences can be due to defect in innate or acquired immune systems. Intrinsic responses in some patients (impaired Innate host defenses) are less effective than in others. Variability in neutrophil function or in mediator production by macrophages is regularly found among all patients.

On the contrary, impairment in the acquired immune system is evidently associated with high rates of SSI. Hypoxemia and shock are positively correlated with SSI, for example in patients with history of trauma. Transfusion appears to be immunosuppressive. Correspondingly, hypo-albuminemia, and malnutrition, chronic illnesses are important contributing factors. Other important variables that impair host response are Hypothermia and Hyperglycaemia. Medications such as corticosteroids could also adversely impact the host immune system and thus lead to high rates of SSI.

Microbial factors of importance in the development of infection

Size of the inoculum and nature of the microbe

One of the primary determinants of onset of infection is the size of the microbial inoculums, which or bacteria is expressed in terms of colony forming units (CFU). Two major reservoirs of microbes exist that can form the initial inoculums leading to infection in surgical patients.

Host endogenous micro flora Microbes within the external milieu, which often represents the nosocomial environment for hospitalized individuals.

The essential component in the SSI is the pace at which pathogens multiply. Microbial division is dependent on ambient temperature and oxygen concentration (varying from one microbial species to another), supply of nutrients and intrinsic properties which decides the rate at which division can occur in the most favourable conditions. The term "virulent" is applied to pathogenic microbes that can lead to disease, and which can consistently produce severe infection. Certain microbes though not inherently virulent, acquire virulence when there is disruption in or suppression in host defenses.

Virulence and pathogenicity

Refer to the ability of a microbe to produce disease or tissue injury. 'Pathogenicity' is generally employed to refer to the ability of a microbial species to produce disease 'Virulence' is applied to the same property in a strain of microorganism. Virulence of a microbe is sum total of several determinants, as listed below:

Adhesion

Invasiveness

Toxigenicity

Communicability

Bacterial products and appendages

Infecting dose

Route of infection

Rate of microbial proliferation

If a surgical site is contaminated with more than 10^5 microorganisms per gram of tissue, quantitatively, it has been shown that the risk of SSI is significantly higher. However if a foreign material is present at the site for example: 100 staphylococci per gram of tissue introduced on silk sutures, the dosage of the contaminating microorganisms required to produce the infection is much lower. Many

factors govern the development of SSI and the microbial factors have been discussed above. Mere presence of bacteria in the surgical site does not cause infection in lieu of the solid host defense mechanisms.

When an organism enters (e.g. as at a surgical site or via an indwelling catheter), host defence mechanisms act and try to remove the pathogens and if the initial barrier fails, additional host defenses are recruited to the portal of entry of the microbes.

Defense Mechanisms

These mechanisms consist of the following factors:

Physical barriers and host microflora

Sequestration mechanisms

Immune mechanisms

Physical barriers and host micro flora[5]

The primary defence against all kinds of pathogens is the anatomical barrier. The most important part of any barrier is that each of them possesses an integument/epithelial or respiratory, gut, uro-genital endothelial surface. The barriers effectively protect from the entry of pathogens into areas that are under normal circumstances sterile. The primary challenge to the surgeon is the endogenous micro-flora, which usually forms the major source of SSIs. More important than that is the

fact that these microbes are easily amenable to be sterilized before barrier disruption takes place by the usage of topical microbicides, intra-luminal antiseptics, and or AMP (anti-microbial prophylaxis), either topical or systemic.

Integument, the first physical barrier, is the largest organ in the human being, being expansively spanned and having a dry weight of about 4 kilograms. Integument also acts as the largest interface between the external microbial milieus and the internal milieu of the host. It provides for much more than a mechanical barrier. The healthy skin possesses bactericidal activity to which the presence of high concentration of salt in the drying sweat, the sebaceous secretions and the long chain fatty acids and soaps contribute.

Though the skin frees itself readily of micro flora deposited on it (transient), its reactions are different to the bacterial flora normally resident on it. Even washing and application of disinfectants do not easily remove resident flora.

Hence it is important that the resident flora is kept to a minimum before deliberate wounding or breach of this vital barrier occurs.

Infection at the surgical site may also occur as a consequence of microbial invasion at other barrier sites and subsequent dissemination and seeding at the surgical site.

Sequestration mechanisms[6]

In any tissue, resident and recruited host defences attempt to kill and eradicate all microbes. It is further augured by influx of inflammatory fluid that contains amongst many other substances, fibrin which blocks bacteria in the extra cellular space.

Sequestration mechanisms on the other hand also acts as a double edged sword, on one hand limiting the focus of infection and on the other by preventing resident and recruited defence mechanisms to reach the site of inflammation by walling off the later.

This invariably leads to the formation of the abscess. However sequestration is in a way an advantage as it converts grave infections into chronic type which can be tackled subsequently.

Immune mechanisms[7]

Immune mechanisms are of two types: humoral immunity and cellular immunity. In Humoral immunity its elements circulate in the body in the form of proteins, and includes 2 types: Antibodies and Complement

Cellular immunity comprised of the T lymphocytes receives the initial load of antigens (microbial or otherwise) and phagocytose them for further killing. Further killing of the microbes is usually via lysis as governed by multiple mechanisms including the bacterial degradation by vacuolation and by release of oxygen free radicals. Cellular immunity generally is triggered within 2 to 4 hours of microbial inoculation (in this instance, after the incision is made) into the tissues and it is this lag period that a surgeon should guard and cover by means of antimicrobial prophylaxis (AMP) whenever justified.

The cellular immunity is influenced by multiple factors, innate or acquired. Cytokines, like the interferon, tumor necrosis factor, and interleukin offer innate co-ordination of cellular immunity. They further aid to regulate the host defenses. The acquired factors governing cellular immunity include the previous exposure of antigenic material (as in immunization), immunocompromised status like as in diabetes mellitus, malnutrition, AIDS, and long term steroid therapy.

Forms of healing

Surgeons customarily divide types of wound healing into. First intention (primary) healing occurs when tissue is cleanly incised and re-approximated and repair occurs without complication.

Second intention (secondary) healing occurs in open wounds through the formation of granulation tissue. Granulation tissue is the red, granular, moist tissue that appears during healing of the open wounds. Microscopically it contains new collagen, blood vessels, fibroblasts, and inflammatory cells, especially macrophages. Covering of this tissue is then followed by spontaneous regression of the epithelial cells. Most infected wounds and burned tissue heal by the way of second intention.

The nature of repair

In a broader sense, the nature of repair has been depicted schematically. As this topic is centered on surgical sites and infections, only healing of a surgical incision is described here. The narrow incisional space immediately fills with clotted blood containing fibrin and blood cells; dehydration of the surface clot forms the well known scab that covers the wound.

Within 24 hours, neutrophils appear at the margins of the incision, moving towards the fibrin clot. The epidermis at its cut edges, thickens as a result of mitotic activity of the basal cells, and within 24 hours to 48 hours, spurs of epithelial cells from the edges both migrate and grow along the cut margins of the dermis, depositing basement membrane components as they move. They fuse in the midline beneath the scab, thus producing a continuous, albeit, thin epithelial layer.

By day 3, macrophages largely replace neutrophils. The incision space is progressively invaded by Granulation tissue. Collagen fibers reach the margins of the incision, but initially they are vertically positioned and do not bridge the incision. As the proliferation of epithelium continues, the epidermal covering layer becomes thickened.

By day 5, the incisional space is filled with granulation tissue. Maximum neovascularization is evident. Collagen fibrils start to bridge the incision and become more abundant.

There is sustained proliferation of fibroblasts with accumulation of collagen during the 2^{nd} week. The edema, augmented vascularity and leukocytic infiltration, largely disappear. By this time, blanching starts, due to regression of vascular channels followed by increased accumulation of collagen within the incisional scar.

Growth factors and cytokines affecting various steps in wound healing

Monocyte chemotaxis	PDGF, FGF, TGF-beta
Fibroblast migration	PDGF, EGF, FGF, TGF-beta, TNF, IL-1
Fibroblast proliferation	PDGF, EGF, FGF, TNF
Angiogenesis	VEGF, Angiogenesis, FGF
Collagen synthesis	TGF-beta, PDGF
Collagen secretion	PDGF, FGF, EGF, TNF, (TGF-beta inhibits)

PDGF- platelet derived growth factor, EGF- epidermal growth factor, FGF- fibroblast growth factor, TNF- tumor necrosis factor

Impaired healing8,9

Of the many causes incriminated in defective wound healing, tissue hypoxia resulting from cardiopulmonary diseases, peripheral vascular diseases, and malnutrition, diabetes mellitus and chronic inflammatory disorders are a major cause. A prior search into these problems is a must before surgery is undertaken.

The repair process is influenced by many factors including. The tissue environment and the extent of the tissue damage. The intensity and duration of the stimulus. Conditions that inhibit repair, such as the presence of foreign bodies or inadequate blood supply. Various diseases that inhibit repair (diabetes in particular) and treatment with steroids.

MICROBIOLOGY

Organisms most frequently isolated from the wound are to be given priority and a full knowledge of its growth, characteristics and antimicrobial agent sensitivity patterns is necessary to tackle them effectively.

A further knowledge of host microflora is essential as some of them often represent the initial inoculums of pathogens when damage or breach of a physical barrier occurs. Knowledge of this to the surgeon is a must as one of the parameters of prevention of SSIs is predicted on reducing the number of resident microbes and prevent barrier disruption using topical microbicidal or intra-luminal antiseptics and or antimicrobial agents, plus systemic antimicrobial agents to reduce the threat of invasion of these resident microflora and their proliferation once wounding, either deliberate (surgical incision) or traumatic occurs.

The following table depicts the common microflora of the intact skin[4]

Region	Microbes[#]	Quantity
Skin (all areas)	Acinetobacter Brevibacterium Corynebacterium* Micrococcus Pityrosporum Propionibacterium* Staphylococcus aureus and Epidermidis* Streptococcus (non-enterococcal)	
Skin (infraumbilical)	Candida Streptococcus fecalis, Escherichia coli	
# Potential pathogenic organisms * These organisms are also found in infra- umbilical region		

Bacteria of surgical interest

Bacteria of surgical interest, which are encountered in SSIs frequently, are discussed here. Bacteria that cause SSIs frequently are staphylococci, streptococci, and the enterobacteriaceae. A few of them are described here.

Staphylococcus: They are gram positive, either aerobic or facultative anaerobic cocci with staphylococcus aureus being the most common species. The organism is found in the nose and throat of 30% to 50% of healthy carriers. Antibiotic resistant hospital strain of S. aureus may be carried in the nose, throat and on the hands of hospital staff.

Staphylococcus aureus: elaborates toxins like hemolysin, leucocidin, enterotoxin, hyaluronidase, fibrinolysin, nuclease, lipase, and protease. In addition, non-toxic substances like coagulase, which help in the invasion of bacteria, are also produced. Today, multi-drug resistant Staphylococcus aureus is probably the most severe cause of SSI, which has to be dealt with.

Staphylococcus albus (S. epidermidis), generally not considered a pathogen but an agent of opportunistic infection which is found on the skin, in air, water and dust has also acquired resistance to multiple drugs (multi drug resistant S. epidermidis – MRSE) in view of wide spread use

of AMAs and increasing incidence of immune compromised status in surgical patients.

Streptococcus: They are Gram-positive cocci, either aerobic or facultative anaerobic or even anaerobic.

Streptococcus pyogenes: They are facultative anaerobic organisms. One of the commonest bacterial pathogen of humans, it is an invasive microorganism and secretes two distinct hemolysins (streptolysin O and S) and several other products which aid in invasion. The hyaluronidase and streptokinase produced by most strains of S. pyogenes are responsible for spreading cellulitis. When an abscess forms, the pus is watery and often blood stained due to the action of these enzymes.

Streptococcus fecalis (Enterococci): This is found as a common organism in the human intestine. It causes suppurative SSIs and urinary tract infections.

Peptostreptococcus putridus (Anaerobic Streptococcus): This normally inhabits the mouth, intestine, and vagina. Anaerobic streptococci have also been isolated from puerperal sepsis. Symbiotic activity of Peptostreptococci, E. coli and Bacteroides cause synergistic gangrene/necrotizing cellulitis with widespread involvement of deeper tissues (Meleny's gangrene), in postoperative patients (patients subjected

to surgeries on the colon and appendix), and in critically ill patients. Infection of the surgical site leads on to crepitant cellulites, which has to be differentiated from the crepitant cellulitis caused by clostridia species.

Enterobacteriaceae: This is a family of inter-related gram negative bacilli isolated from the intestinal tract and has pathogenic members like Escherichia coli, Klebsiella, and Proteus, commonly found as intestinal commensals. These commensals usually cause urinary tract infections, SSIs, infections of the biliary tract in the post operative period

Escherichia coli: These non capsulated, motile, aerobic, and facultative anaerobic bacilli are found as intestinal commensals in humans. The organism enters the intestine shortly after birth and persists throughout one's life. These bacilli are concentrated in the ileo-caecal region and diminish up to the duodenum proximally and rectum distally. These bacteria serve a useful function by synthesizing B-complex vitamins and also by suppressing the growth of certain proteolytic organisms.

However these are one among the common organisms that cause a myriad of infections including SSIs, appendicitis, cholecystitis, peritonitis, and urinary tract infections.

Klebsiella: These are gram-negative aerobic (or facultative anaerobic), non-motile, and capsulated bacilli found on mucous surfaces of the upper respiratory tract, intestines and the genitourinary tract. Along with other gram-negative bacilli, they have assumed increasing importance as a cause of nosocomial infections. They are known to cause endocarditis, septic thrombophlebitis, septicemia, wound infections, crepitant cellulites, and myonecrosis. Klebsiella pneumoniae causes severe pneumonia in debilitated patients.

Enterobacter aerogenes: These organisms, found in the intestine are closely related to Klebsiella in their morphology and though less pathogenic than former, are demonstrated as common isolates in nosocomial sepsis.

Proteus: The organisms of the Proteus group (Proteus vulgaris, P. mirabilis, P. morganii, and P. rettes) are usually found on rotten meat and vegetables, sewage, soil, manure, and in feces of humans and animals. Though of considerable low pathogenecity, these organisms are notorious isolates in cultures from nosocomial infections like that from surgical sites, bed sores, burned wounds and urinary tract.

Pseudomonas aeruginosa: These are actively motile, gram-negative, and strictly aerobic bacilli found in the intestines of humans and animals, sewage, water and soil. As frequently secondary invaders, they produce

inflammatory and suppurative lesions in humans; the purulent discharge produced usually being greenish-blue in color with a characteristic sweetish odor. Resistance of these organisms to the common groups of antimicrobial agents used in now frequently met with in hospital settings. As such, owing to its resistance, it has become one of the front-runners amongst drug resistant bacteria to cause fulminant septicemia owing to secondarily infected burn wounds, SSIs, urinary tract infections and respiratory infections in mechanically ventilated patients.

Clostridium: These are gram-positive, spore-bearing, anaerobic, saprophytic bacilli, found in the intestines of cattle, horse and humans. Their spores are found in the soil, dust, and sewage contaminated with human and animal excreta.

They are fastidious anaerobes, requiring a low redox (reduction-oxidation) potential to grow and to initiate conversion of spores to vegetative, toxin producing pathogens.

Such an environment is seen in tissues with diminished blood supply, muscle injury, foreign body implants and in mixed infections with pure aerobic bacteria.

Manifestations of clostridial infections include tetanus, gas gangrene, or botulism depending upon the species involved. The gold standard of sterility of a surgical instrument or that of a sterile atmosphere and equipments in the operating suite is the absence of growth of C. tetani on serial cultures taken from these places. Bacterial spores of C. tetani are also used as indicators of effective sterilization of surgical apparel by dry heat or by autoclaving.

In the normal oral cavity, anaerobes out number aerobes by 10:1. And in the normal colon, by 1000:1. When trauma, disease or surgical procedure disrupts the mucous membrane barrier, these bacteria can invade the contaminated tissues and may cause infection.

Bacteroides: They are the sparse normal flora of the mouth, respiratory tract, intestine and vagina. Species causing infections in human beings are Bacteroides fragilis and B. melaninogenicus, the latter producing black colonies on culture media. Foul smelling exudates from wound site with no growth on aerobic cultures are often due to infections with these anaerobic organisms.

Though higher virulence is rare with these organisms, multiple organ abscesses with Bacteroides as a result of septicemia are quite common in immune-compromised patients.

Nocardia are traditionally considered as transitional forms between bacteria and fungi. Many species of Nocardia exist and all are aerobic. All strains are gram-positive and some strains, acid fast in addition. Increasing interest in these organisms is due to the fact the serial SSI outbreaks, isolation Nocardia have come to light in the west.

In the event of a SSI occurring, it becomes a dilemma as to decide the nature of infecting organism and the nature of treatment to be instituted against the same. Over a period of time, repeated occurrences of infections and repeated isolates have indicated predilection of certain organisms towards particular surgical sites. Knowledge of the same would be helpful in commencing empirical treatment till culture and sensitivity pattern reports are available.

CLINICAL FEATURES OF SURGICAL SITE

INFECTIONS

Superficial/deep SSIs

SSIs usually appear between the fifth and tenth days of surgery, but they appear as early as the first day or even years later. The first sign usually is fever, and fever in an operated patient warrants inquiry of the wound. The patient may complain of pain at the surgical site. The surgical site rarely appears severely inflamed, but edema may be obvious because the skin sutures appear tight.

Palpation of the surgical site may disclose an abscess. The rare infection deep to the fascia may be difficult to recognize. In doubtful cases, one can carefully open the wound in suspicious area using meticulous sterile technique. If no pus is present the wound can be closed immediately with skin tapes. Cultures, even of clean wounds that are successfully re-closed during the time of surgery are often positive.

Organ/space SSIs

Intra-abdominal abscess can be classified into two types: Intra-peritoneal abscess. Retroperitoneal/retro-fascial abscesses. An intraperitoneal abscess should be suspected whenever there is a

predisposing condition. Fever, tachycardia and pain may be mild or absent, especially when the patient is on antibiotics or has received them perioperatively. Some patients with deep seated, small collection may be well as others but there may be persistent mild fever. Prolonged ileus, or delayed recovery of GI functions, in a patient recently subjected to an abdominal surgery, coupled with raising leucocytes, or nonspecific radiological abnormality provides the initial clue.

Differential diagnosis of SSIs

Include all other causes of postoperative fever Physiological postoperative fever - due to catabolic process induced by the stress of surgery, or the disease process or both. Urinary tract infections - as differentiated by history of burning micturation, pain abdomen, urgency, frequency, fever with or without chills and rigors and rarely hematuria. Confirmation of urinary tract infection is by microscopic examination of urine and gram's staining. If warranted a culture and appropriate sensitivity pattern study is also undertaken.

Respiratory tract infections - as differentiated by history of postoperative fever, malaise, dyspnea, and productive or non-productive cough. Confirmation warrants a chest radiograph and sputum analysis by gram's staining

Intravascular catheter infections - as differentiated by history of irritation at the catheter site, pain and redness at the site or extending in a linear fashion along the catheterized vessel, postoperative fever, sometimes with chills and rigors. Confirmation necessitates culture of blood from the site of catheterization, or removing the device and remitting it to the culture directly.

Blood stream infections and sepsis syndrome - as differentiated by history of postoperative fever or cold clammy extremities and that of shock. Rigors may precede the clinical presentation and may continue well into the course of the disease. Jaundice is due to hepato-cellular damage. Endotoxin mediated peripheral circulatory collapse causes dysfunction of vital organs. Patients may lapse into coma due to profound shock and failure at prompt resuscitative and supportive measures.

Most often, the offending sites are the gastrointestinal or the urogenital tracts. Wound dehiscence - as differentiate by a history of sudden acute pain with a give way sensation at the operated site and a sudden relief from the acute pain with or without fever and a serous or sero-sanguinous (pink) discharge from the surgical site. Evisceration of the abdominal contents may also be complained of. Examination concludes the diagnosis in the form of evident gaping of the

approximated structures or sometimes as a defect under an intact cutaneous covering.

Prevention and Treatment of Surgical Infections

Appropriate Use of Antimicrobial Agents

Prophylaxis includes administration of antimicrobial agent or agents before the initiation of particular types of operative procedures in order to reduce the number of pathogens from entering the tissue or body cavity.

Empiric therapy consists of the administration of antimicrobial agent or agents when the possibility of operative site infection is high, depending on the underlying disease process for example perforated appendicitis, or when extensive contamination of the site during the operation has occurred for example inadequate preparation of bowel or spillage of contents of colon. Conditions in which the risk of infection is significant because of intra-operative findings, prophylaxis is merged with empiric therapy.

Invariably, empiric therapy should ideally be stopped as soon as possible following microbiologic data, in the absence of positive cultures along with improvements in the clinical condition of the patient. Hence empiric therapy should be restricted to a short duration, 3 to 5 days.

Antimicrobial agents are chosen according to the sensitivity of microbes which are present at the operative site, based on the understanding of host microflora. In case of patients undergoing elective resection of colon should ideally undergo mechanical preparation of the bowel plus receive intraluminal agents directed against aerobes and anaerobes (e.g.neomycin + metronidazole); immediately prior to creating the incision, the patient should be administered a single dose of any one of a large number of intravenous agents with a similar spectrum of activity

Surgical antimicrobial prophylaxis (AMP) pertains to a brief duration course of an antimicrobial agent started just before the surgery begins. The aim of AMP is not to sterilize tissues, but is to reduce the microbial load of intra-operative contamination at a crucially time to an extent that will not overwhelm the host defense mechanisms.

AMP is not related to the prevention of SSI caused by post operative contamination. AMP delivery in modern surgical practice is mostly intravenous but not necessarily the only route that must be employed.

To attain maximum benefits of AMP, four principles should be followed:

1. AMP agent should be used for all procedures or classes of procedures in which its use has demonstrated to reduce the rate of SSI on the basis of evidence on clinical trials or for those surgeries following which incisional or organ/space SSI would present with a disaster.
2. AMP agent of choice is one that is safe, has bactericidal property, is inexpensive, and had a broad spectrum that would cover most of the intra-operative contaminants of the surgery.
3. The initial dose of antimicrobial agent (AMA) should be timely infused so such that bactericidal concentration of the agent is reached in the serum and tissues by the time the skin is incised.
4. Therapeutic levels of the AMA should be maintained in both serum and tissues throughout the surgical procedure and continuing for a few hours after the closure of incision in the operating room. As clotted blood develops in all wounds, achieving therapeutic tissue concentrations of AMAs are vital.

SELECTION OF AMP AGENT

Selecting AMP, based on indications is guided by the postoperative class of the surgery as detailed previously (clean, clean contaminated, contaminated or dirty). However, AMP is administered ideally preoperatively in anticipation of the postoperative class of the surgery.

There are no clear cut guidelines as to when the AMP should be employed, but in general, AMP should be used for all "clean contaminated surgeries" (that is when a hollow viscus is opened under controlled conditions). The concept of AMP is not indicated in the "contaminated" or "dirty" surgeries, as in such operations, patients are frequently receiving therapeutic AMAs perioperatively for established infections.

The three main indications for employing AMP in "clean" surgeries are as follows:

Insertion of intravascular prosthetic material or prosthetic joint for any operation in which an incisional or organ/space infection would predispose to catastrophic risks (e.g. cardiac surgeries, pacemaker implant surgeries, prosthetic arterial graft placement, neurosurgical operations, and when a prosthetic mesh is placed for hernioplasty). Many have also recommended AMP for all surgeries on the breast.

There are few more sensitive criteria in choosing an AMA for AMP:

Such an AMA should not be highly toxic and should be largely devoid of adverse effects. Such an AMA should not be “first line” AMAs for the treatment of established infection. This is important because resistance may develop to the administered AMAs rapidly, and such agents used frequently in prophylaxis are likely to lose their effectiveness for later treatment. Such an AMP should be capable of combating the anticipated organisms in a selected procedure.

CHOOSING THE AMA

Not many AMAs fit the bill to be ideal choices for AMP but the front runners, as the AMAs of choice for AMP are the cephalosporins. Cefazolin, a first generation cephalosporin, which adequately covers gram-positive and gram-negative organism has been considered an adequate and safe AMA for prophylaxis in all “clean” and many “clean contaminated” procedures.

However, second generation cephalosporins like the cefoxitin or cefotetan, which have an additional activity against anaerobes, may be needed to be employed in clean contaminated surgeries of the colorectum or gynaecological procedures.

If the patient is allergic to penicillins or cephalosporins, aztreonam clubbed with clindamycin or metronidazole for coverage against anaerobic bacteria seems to be a safe alternative.

Vancomycin created a rage when first introduced as an AMA. It was virtually effective against all resistant gram-positive organisms and probably still is. However, this drug exhibits considerable toxicity and is expensive and hence does not suit the bill for being an AMA of choice for AMP.

Other contenders for AMP agents of choice include the flouroquinolones like the ciprofloxacin and amoxyicillin-clavulanic combinations.

None of the above have been as extensively researched and subjected to trials as the cephalosporins and to conclude on these agents at this point in time may be erroneous.

Guidelines for the administration of AMP are as follows:

A single therapeutic dose of the selected AMP agent is to be given half an hour prior to the skin incision. This to achieve maximum therapeutic concentrations when the vital integumental barrier is breached and to maintain the same level of drug concentration till the procedure is complete and a few hours beyond.

Additional doses of the AMP agent may be unnecessary except in surgeries extending to beyond 2 hours or those that require large volumes of resuscitative fluids (because of the larger volume of distribution of drug).

Additional dosing of the selected AMP agent may be considered if the total blood loss at the time of surgery or during the preoperative period exceeded 2 L.

Subsequent dosing and the timing of the same is to be guided by the following parameters:

1. Standard therapeutic dose achieving tissue levels in normal patients.
2. The serum half life of the drug administered
3. Approximate MIC 90 values for the anticipated SSI pathogens.

There are few exceptions to these guidelines.

Concerning the dose of the drug, it has been shown that larger doses than that considered therapeutic may be necessary in morbidly obese patients[43].

HAZARDS OF AMP

Antibiotic resistance, perhaps, is the most dreaded of all the hazards of AMP. The knowledge of what might ensue out of this inappropriate is nebulous at best in the current time and trends harbinger towards a catastrophe if similar attitudes and misconceptions on AMP prevail.

Apart from resistance, AMA per se have their own inherent side effects, like drug allergy, toxicity (predicted/idiosyncratic), intolerance, carcinogenic/mutagenic effects and drug induced diseases. Hence AMP needs to be employed only when the advantages of its use far out weigh the risk of hazards due to its usage.

However, one has to remember that AMP cannot and is not intended to eliminate bacteria.

Use of multiple AMAs

Increases the dug interactions

Diminishes the effectiveness in the long run by promoting the emergence of resistant strains Conversely increasing the cost of managing surgical patients and SSIs.

It is worth while here to reiterate that AMP should be given only when a significant rate of infection is encountered without prophylaxis or when the consequences of infection would be disastrous, as with placement of vascular, cardiac, or joint prosthesis.

MANAGEMENT OF SSIs[9, 14]

Superficial/deep SSIs

The basic treatment of established wound infection is to open the wound and allow the wound to drain. Antibiotics are not necessary unless the infection is invasive, manifested by a surrounding zone of soft tissue inflammation (erythema and edema), or features suggestive of cellulitis (> 2 cm beyond the incision site).

Culture should be performed to locate the source and prevent further infection in other patients, to gain a preview of bacterial flora in case other infections develop deep to the wound or in case existing infection becomes invasive and to select preoperative antibiotics in case the wound must be entered again.

Organ/space SSIs

Treatment consists of prompt and complete drainage of the abscess, control of the primary cause, and adjunctive use of effective antibiotics. Depending upon the abscess site and the condition of the patient, drainage may be achieved by operative or non-operative methods. Per-cutaneous needle/catheter drainage is preferred method for single, well localized, superficial bacterial abscesses that do not have fistulous

communications or contain solid debris. CT scan/ultrasonographic delineation aids in placement of needle/catheter for drainage of the abscess, which can be accomplished in a single sitting or multiple sittings with minimum discomfort to the patient.

Prognosis of SSIs[1]

Most superficial/deep SSIs make illness more severe. SSIs correlate positively with death rates but superficial/deep SSIs are not often the cause of death. It, however certainly tilts the scales against successful surgeries and unnecessary to say that, it also tilts the success of the attending surgeon.

Organ/space SSIs are more severe, and serious intra-abdominal abscesses carry mortality rates of about 30% despite adequate management. Deaths are related to the underlying cause, delay in diagnosis, multiple organ failure, and incomplete drainage. Recent studies have established that decompensation of even two major organ systems raise the mortality rates to 50%. Shock is an ominous sign. An untreated, residual abscess is as nearly always fatal.

Reversal of clinical features or an improvement in same within 3 days of intervention often points to a successful treatment. Failure of such pattern warrants immediate revaluation of patient and appropriate management; else condition could become fatal.

SSI SURVEILLANCE

A surveillance system uses epidemiologically sound infection definitions (as elucidated in tables earlier) and effective surveillance methods, stratification of SSI rates according to the risk factors associated with SSI development, and data feedback.

The two primary components of the surveillance are the SSI risk stratification and surveillance methods.

Risk stratification is based on mainly three variables.

Those estimating the intrinsic degree of microbial contamination of the surgical site.

Those that measure the duration of operation.

Those that serve as markers for host susceptibility.

The widely accepted model assessing the first variable has been described in the table. The center for disease control (CDC) has undertaken two classical projects in the form of SENIC project and the NNIS system which have incorporated numerous other variables to the one proposed in the table for SSI risk indices.

SSI surveillance methods are multimodal. Those recommended in the SENIC project and the NNIS system was for in-patients. However, over the last decade and half, day care surgeries are the vogue and hence the surveillance system had to be expanded to encompass discharged patients too.

In-patient surveillance is easy but grossly considered inadequate as a SSI can occur after discharge of the patient after a significant period has elapsed postoperatively. Various studies have documented 12% to 84% of SSIs, occurring post discharge. Post discharge surveillance hence assumes significance if one needs to have an accurate account of the SSI rate and it's mode of presentation. Post discharge surveillance needs serious, dedicated effort from the investigator and can be achieved by the following methods.

Direct examination of the patient's wounds during follow up visits to either the surgical centers or to the physician offices. In the latter circumstances, the attending physician has to promptly alert the investigator apart from treating the patients from symptoms.

Review of medical records of surgery clinic patients.[54]

Patient surveys by mail or telephone or by other electronic media.[55]

Surgeon surveys by similar methods.

It really helps if the attending surgeon is also the primary investigator in SSI surveillance as the lapses in form of case misses or defaults are reduced.

A third, more radical and novel method of SSI surveillance is considered to be the out patient surveillance. Here the investigator surveys all the surgical outpatients as regards SSI. It can be said to be the most unbiased or impartial method of assessing the risk. However, it is quite radical in that a large patient base will have to be created, shifted through, and backed by good database plus a good network between various or perhaps, all surgical centers of the country. Though ideal, outpatient surveillance is cumbersome, hence not used extensively.

METHODOLOGY

Source of data

Patients admitted as inpatients in coimbatore Medical College Hospital for Class I (clean) and Class II (clean contaminated) elective general surgeries between june 2013-june 2014

Calculated sample size: 100

Among 100 patients 50 received loading dose of antibiotics during induction of anaesthesia and 50 received post operative empirical antibiotics. Among 100 patients 75 had clean wound[CLASS I] and 25 had clean contaminated[CLASS II] wound.

Inclusion criteria

Patients who underwent Class I (clean) and Class II (clean contaminated) elective general surgeries in coimbatore Medical College Hospital,coimbatore

Exclusion criteria

Patients with implants or prosthetic material

Patients with Diabetes mellitus

Patients on steroids, chemotherapy or immuno-suppression

Patients with Contaminated and dirty wounds

Method of collection of data:

Details of cases were recorded including history and clinical examination. Routine pre-operative investigations performed in both the groups. The study group received loading dose of antibiotic, Cefotaxime at the time of induction of anesthesia While the control group received antibiotics post-operatively for 72 hours or more. Operative wound was examined on the second, fifth and eighth post-operative day for signs of surgical site infection. Patients from both the study and control groups were compared for final analyses.

RESULTS

The study was conducted on a total of 100 patients aged between 13-88, of which 75 underwent clean general surgical procedures and 25 underwent clean contaminated general surgical procedures in Coimbatore Medical College Hospital, coimbatore from june 2013 to june 2014.

Among the 75 clean surgical cases, 38 received loading dose of antibiotic at the time of induction of anaesthesia and 37 received post-operative empiric antibiotics for 3 or more days.

Among the 25 clean-contaminated surgical cases, 12 received loading dose of antibiotic at the time of induction of anaesthesia and 13 received post-operative empiric antibiotics for 3 or more days.

SEX DISTRIBUTION

TABLE NO-1

SEX DISTRIBUTION IN LOADING DOSE GROUP

	Frequency	Percent	Valid Percent	Cumulative Percent
Female	14	28.0	28.0	28.0
Male	36	72.0	72.0	100.0
Total	50	100.0	100.0	

Of the 50 cases who received loading dose of antibiotics at the time of induction of anaesthesia

72% were males and 28% were females.

GRAPH 1:

SEX DISTRIBUTION IN LOADING DOSE GROUP

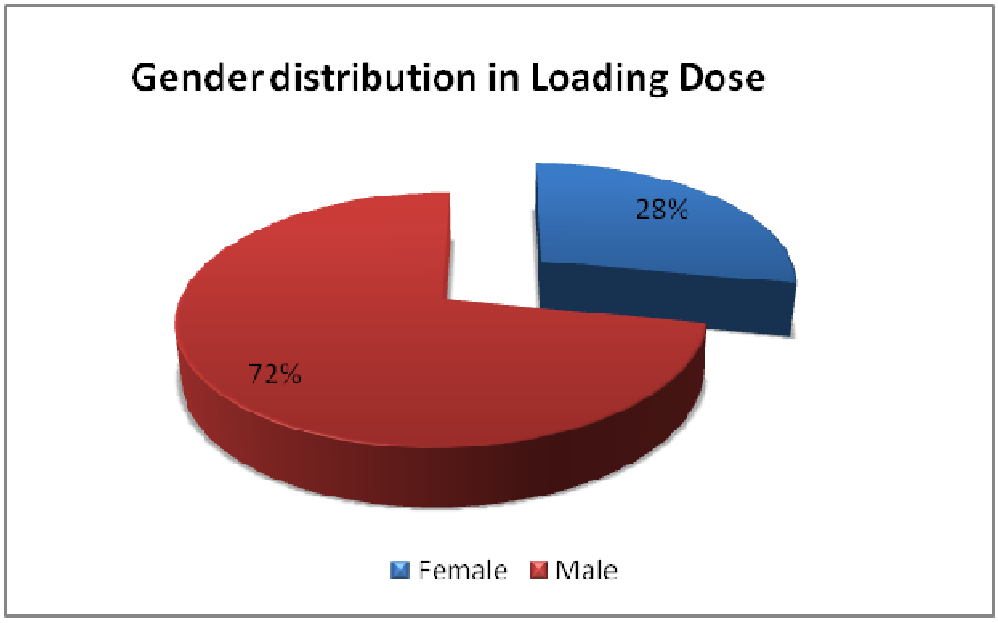


TABLE 2

SEX DISTRIBUTION IN EMPIRIC GROUP

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Female	24	48.0	48.0	48.0
	Male	26	52.0	52.0	100.0
	Total	50	100.0	100.0	

Of the 50 cases who received empiric dose of antibiotics 52% were males and 48% were females.

GRAPH 2

SEX DISTRIBUTION IN EMPIRIC GROUP

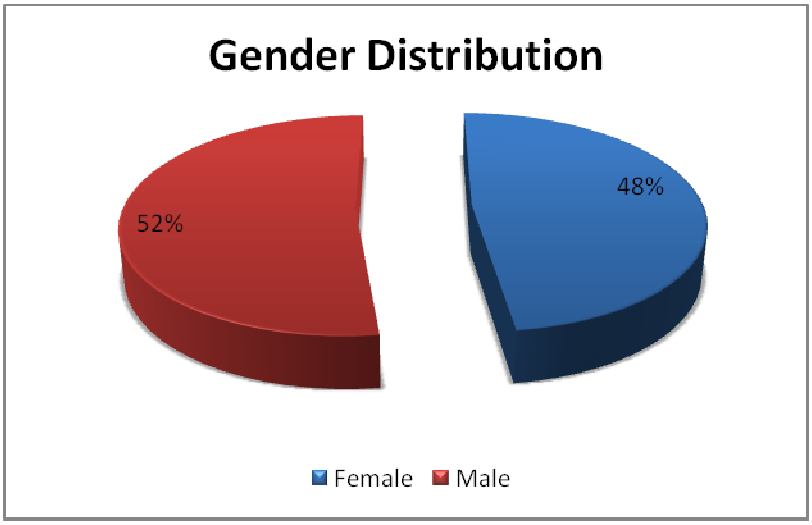


TABLE 3

SEX DISTRIBUTION OVERALL.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Female	38	38	38	38.0
	Male	62	62	62	100.0
	Total	100	100.0	100.0	

Of the 100 cases 62% were males and 38% were females

GRAPH 3 :

SEX DISTRIBUTION OVER ALL

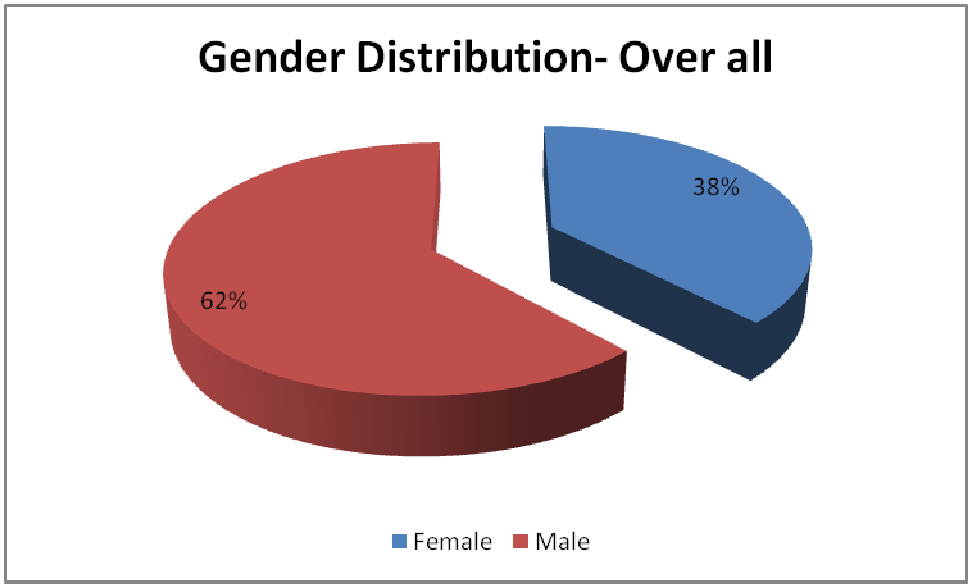


TABLE 4

AGE DISTRIBUTION IN LOADING DOSE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Upto 15 yrs	4	8.0	8.0	8.0
	16 to 30 yrs	11	22.0	22.0	30.0
	31 to 45 yrs	20	40.0	40.0	70.0
	46 to 60 yrs	5	10.0	10.0	80.0
	61 to 75 yrs	9	18.0	18.0	98.0
	Above 75 yrs	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

Among the patients who received loading dose of antibiotics at the time of induction of anaesthesia, the age varied from 13-88 years. The number of patients in the 31-45 years group was highest

GRAPH 4:

AGE DISTRIBUTION IN LOADING DOSE

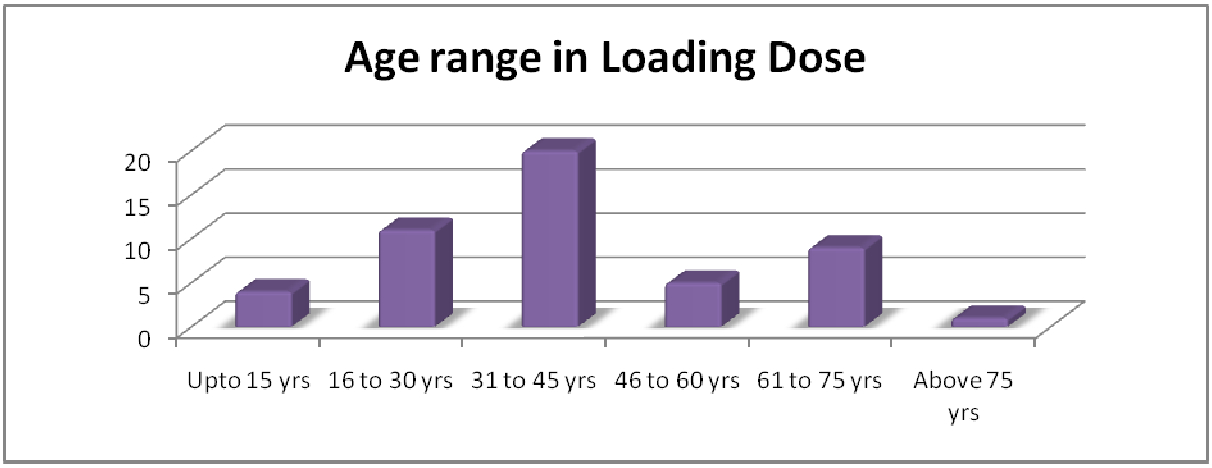


TABLE 5

AGE DISTRIBUTION IN EMPIRIC DOSE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Upto 15 yrs	2	4.0	4.0	4.0
	16 to 30 yrs	12	24.0	24.0	28.0
	31 to 45 yrs	16	32.0	32.0	60.0
	46 to 60 yrs	14	28.0	28.0	88.0
	61 to 75 yrs	6	12.0	12.0	100.0
	Total	50	100.0	100.0	

. Among the patients who received empiric dose antibiotics post-operatively, the age varied from 15-75 years. The number of patients in the 31-45 years group was the highest.

GRAPH 5:

AGE DISTRIBUTION IN EMPIRIC DOSE.

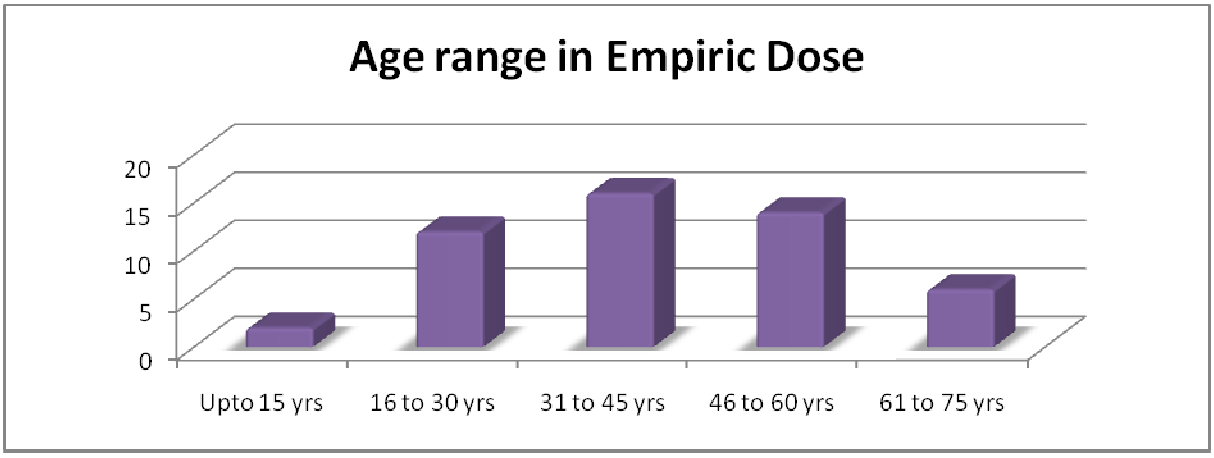


TABLE 6

AGE DISTRIBUTION IN CLASSI[CLEAN WOUND]

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Upto 15 yrs	2	2.7	2.7	2.7
	16 to 30 yrs	15	20.0	20.0	22.7
	31 to 45 yrs	29	38.7	38.7	61.3
	46 to 60 yrs	15	20.0	20.0	81.3
	61 to 75 yrs	14	18.7	18.7	100.0
	Total	75	100.0	100.0	

Among the 75 patients with class 1[clean wound] the number of patients in 31-45 yrs of age group was highest.

GRAPH 6

AGE DISTRIBUTION IN CLEAN WOUND[CLASS I]

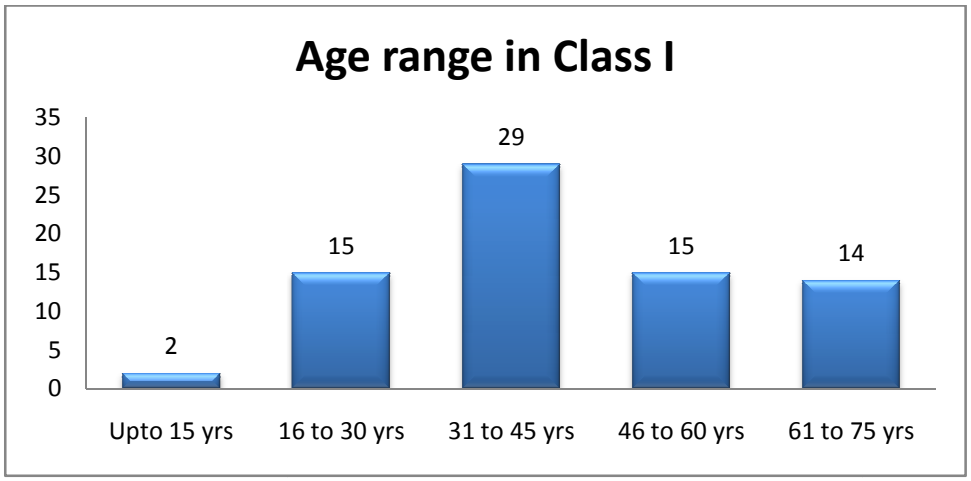


TABLE 7

AGE DISTRIBUTION IN CLASSII[CLEAN CONTAMINATED WOUND]

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Upto 15 yrs	4	16.0	16.0	16.0
	16 to 30 yrs	8	32.0	32.0	48.0
	31 to 45 yrs	7	28.0	28.0	76.0
	46 to 60 yrs	4	16.0	16.0	92.0
	61 to 75 yrs	1	4.0	4.0	96.0
	Above 75 yrs	1	4.0	4.0	100.0
	Total	25	100.0	100.0	

Among the 25 patients with class ii[clean contaminated wound].the number of patients in 16-30 yrs ofe age group was highest.

GRAPH 7:

AGE DISTRIBUTION IN CLASSII[CLEAN CONTAMINATED WOUND]

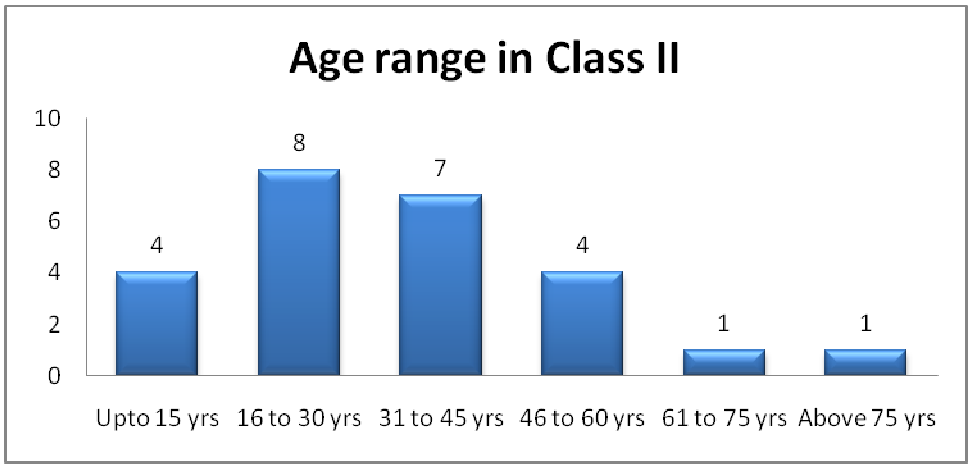


TABLE 8

RESULTS IN CLEAN WOUND WITH SSI

			SSI		
			NIL	PRESENT	Total
LDED	LOADING DOSE		35 50.0%	3 60.0%	38 50.7%
	EMPIRIC DOSE	Count % within SSI	35 50.0%	2 40.0%	37 49.3%
Total		Count % within SSI	70 100.0%	5 100.0%	75 100.0%

Of the 75 patients who underwent class I surgeries, 38 patients received loading dose of antibiotics at the time of induction of anaesthesia. 3 of these patients developed features of SSI .

Of the 75 patients of class I surgery group 37 received empiric therapy for 3 days or more post-operatively, 2 developed features of SSI

TABLE 9

RESULTS OF P VALUE IN CLEAN WOUND WITH SSI

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.187[b]	1	.666		
Continuity Correction[c]	0.000	1	1.000		
Likelihood Ratio	.188	1	.665		
Fisher's Exact Test				1.000	.513
N of Valid Cases	75				

The p value in class I surgeries, when the loading dose group was compared with that of the empiric group, was found to be 0.66 (>0.05). Thus there was no statistically significant difference between the two groups.

GRAPH 8

RESULTS IN CLEAN WOUND WITH SSI

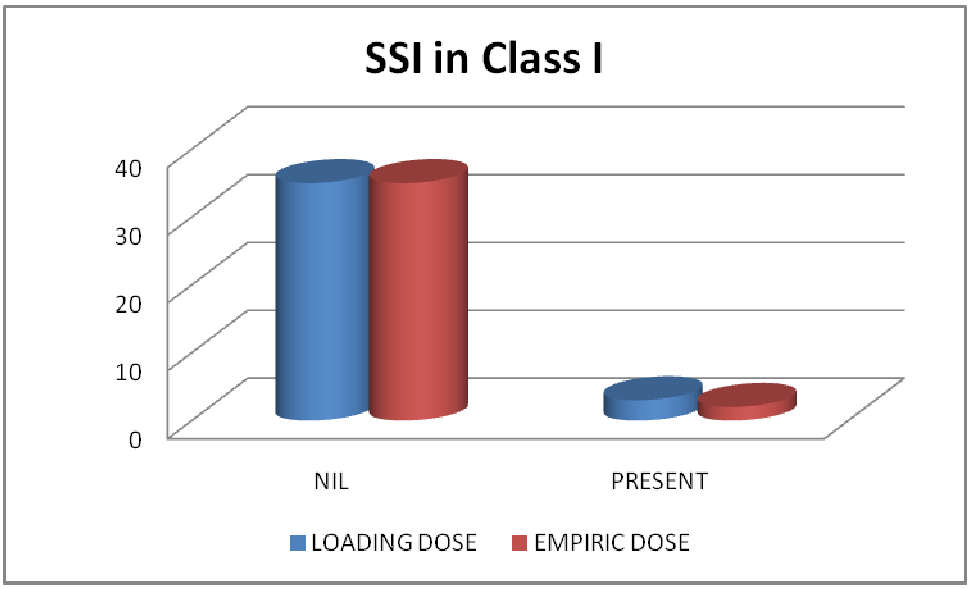


TABLE 10

RESULTS IN CLEAN CONTAMINATED WOUND WITH SSI

			SSI		
			NIL	PRESENT	Total
LDED	LOADING DOSE	Count	11	1	12
		% within SSI	47.8%	50.0%	48.0%
	EMPIRIC DOSE	Count	12	1	13
		% within SSI	52.2%	50.0%	52.0%
Total		Count	23	2	25
		% within SSI	100.0%	100.0%	100.0%

Of the 25 who underwent class II general surgical procedures, 12 patients received loading dose of antibiotics at the time of induction of anaesthesia. 1 of these patients developed features of SSI.

Of the 25 patients 13 Patients received empiric therapy 3 days or more post-operatively, 1 developed features of SSI

TABLE 11

RESULTS OF P VALUE IN CLEAN CONTAMINATED WOUND WITH SSI

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.003[b]	1	.953		
Continuity Correction[c]	0.000	1	1.000		
Likelihood Ratio	.003	1	.953		
Fisher's Exact Test				1.000	.740
N of Valid Cases	25				

The p value in class II surgeries, when the loading dose group was compared with that of the empiric group, was found to be 0.953 (>0.05). Thus there was no statistically significant difference between the two groups.

GRAPH 9

RESULTS IN CLEAN CONTAMINATED WOUND WITH SSI

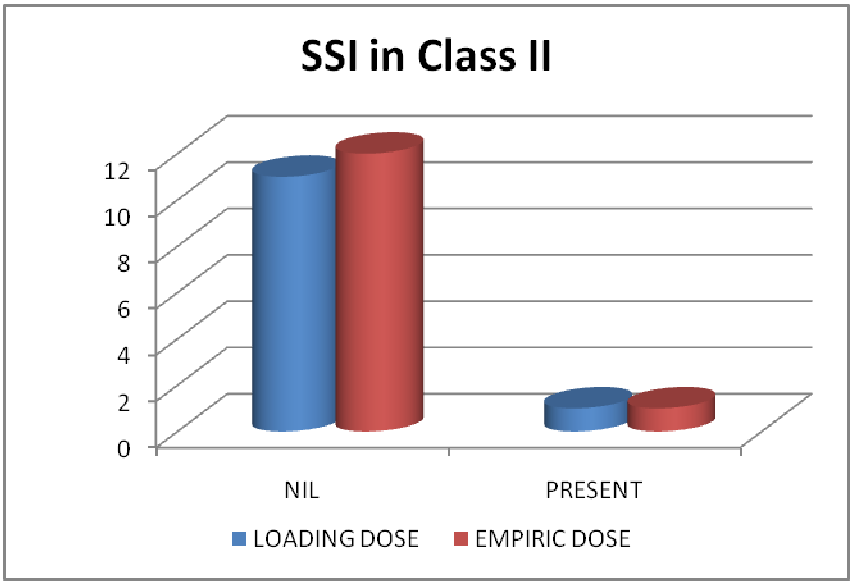


TABLE 12

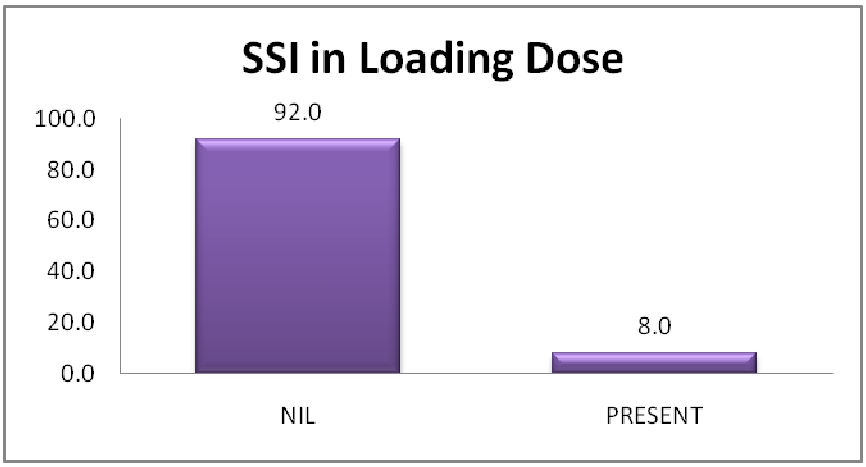
SSI IN LOADING DOSE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	NIL	46	92.0	92.0	92.0
	PRESENT	4	8.0	8.0	100.0
	Total	50	100.0	100.0	

Among the 50 patients who received loading dose 4 [8%] developed SSI.

GRAPH 10 :

SSI IN LOADING DOSE



Erythema-1.

Serous-1.

Purulent-1.

Wound gaping-1.

TABLE 13

SSI IN EMPIRIC DOSE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	NIL	47	94.0	94.0	94.0
	PRESENT	3	6.0	6.0	100.0
	Total	50	100.0	100.0	

Among the 50 patients who received empiric dose 3[6%]developed SSI.

Erythema-2.

Serous-1.

GRAPH 11:

SSI IN EMPIRIC DOSE

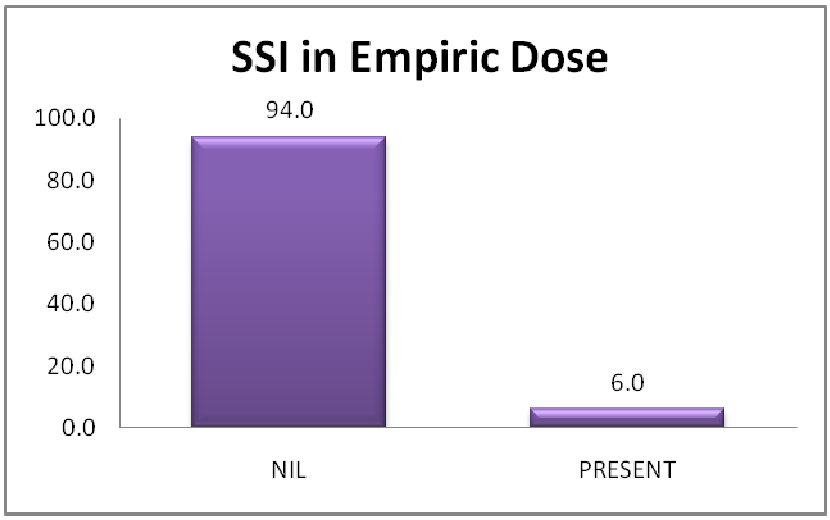


TABLE 14

SSI WITH DOSE-OVER ALL

			SSI		
			NIL	PRESENT	Total
LDED	LOADING DOSE	Count	46	4	50
		% within SSI	49.5%	57.1%	50.0%
	EMPIRIC DOSE	Count	47	3	50
		% within SSI	50.5%	42.9%	50.0%
Total		Count	93	7	100
		% within SSI	100.0%	100.0%	100.0%

Among 100 patients ,50 patients who received loading dose,4 presented with SSI.

50 Patients who received empiric dose 3 presented with SSI.

Thus it was seen that the 4 out of the 50 patients who received a loading dose of antibiotics at time of induction of anaesthesia developed surgical site infections.

3 of the 50 patients who received empiric doses of antibiotics post-operatively developed surgical site infections

TABLE 15

RESULTS OF P VALUE IN SSI WITH DOSE OVER ALL

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.154[a]	1	.695		
Continuity Correction[b]	0.000	1	1.000		
Likelihood Ratio	.154	1	.695		
Fisher's Exact Test				1.000	.500
N of Valid Cases	100				

The p value between these two groups was found to be 0.695 (>0.05).

Hence there was no statistical difference between the two groups

GRAPH 12:

SSI WITH DOSE –OVER ALL

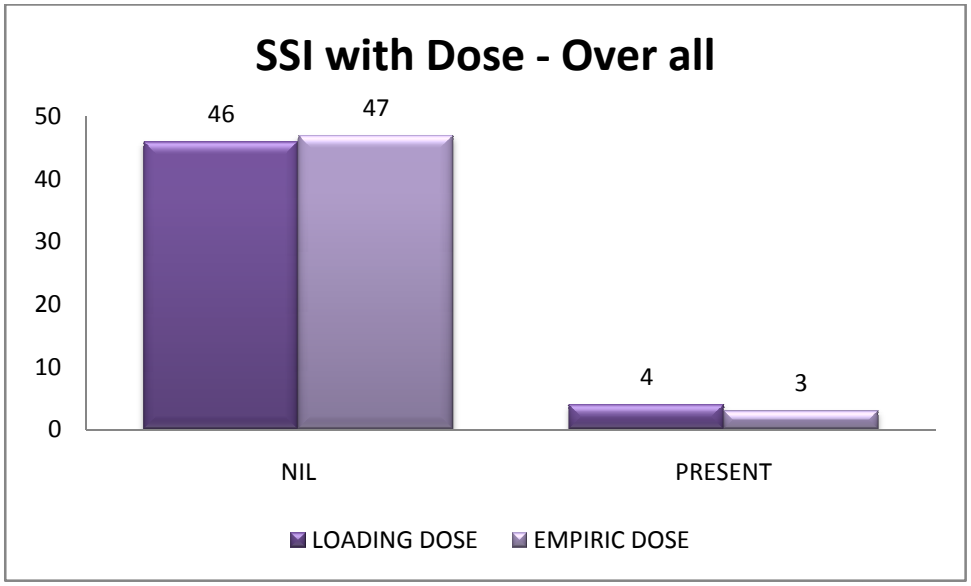


TABLE 16

RESULTS WITH TYPE OF WOUND IN LOADING DOSE

			Typeofwound		
			CLEAN	CLEAN CONTAMINATED	Total
SSI	NIL	Count	35	11	46
		% within Typeofwound	92.1%	91.7%	92.0%
	PRESENT	Count	3	1	4
		% within Typeofwound	7.9%	8.3%	8.0%
Total		Count	38	12	50
		% within Typeofwound	100.0%	100.0%	100.0%

GRAPH 13 :

RESULTS WITH TYPE OF WOUND IN LOADING DOSE

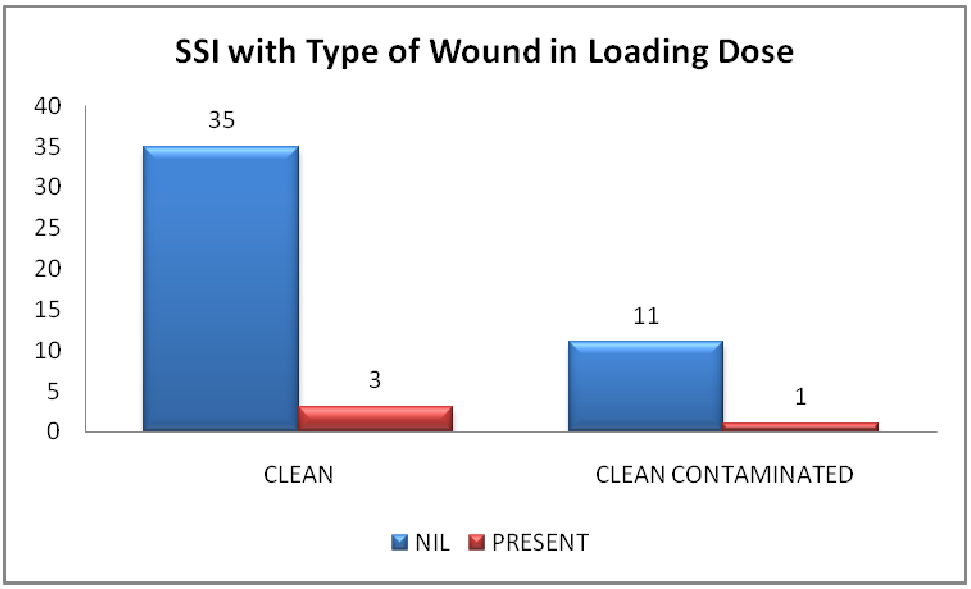


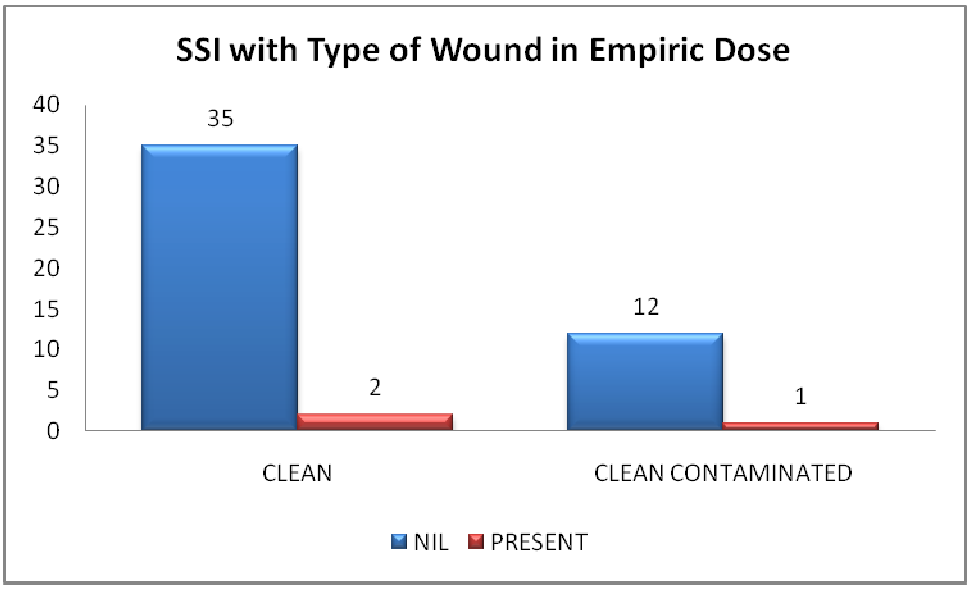
TABLE 17

RESULTS WITH TYPE OF WOUND IN EMPIRIC DOSE

			Type of wound		
			CLEAN	CLEAN CONTAMINATED	Total
SSI	NIL	Count	35	12	47
		% within Typeofwound	94.6%	92.3%	94.0%
	PRESENT	Count	2	1	3
		% within Typeofwound	5.4%	7.7%	6.0%
Total		Count	37	13	50
		% within Typeofwound	100.0%	100.0%	100.0%

GRAPH 14:

RESULTS WITH TYPE OF WOUND IN EMPIRIC DOSE



TYPE OF WOUND DISTRIBUTON.

Among 100 patients class 1[clean wound]-75

Class 11[clean contaminated wound]-25.

TABLE 18

TYPE OF WOUND DISTRIBUTION IN LOADING DOSE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	CLEAN	38	76.0	76.0	76.0
	CLEAN CONTAMINATED	12	24.0	24.0	100.0
	Total	50	100.0	100.0	

Among 50 patients who received loading dose clean wound were 38[76%] and clean contaminated wound were 12[24%].

GRAPH 15:

TYPE OF WOUND DISTRIBUTION IN LOADING DOSE

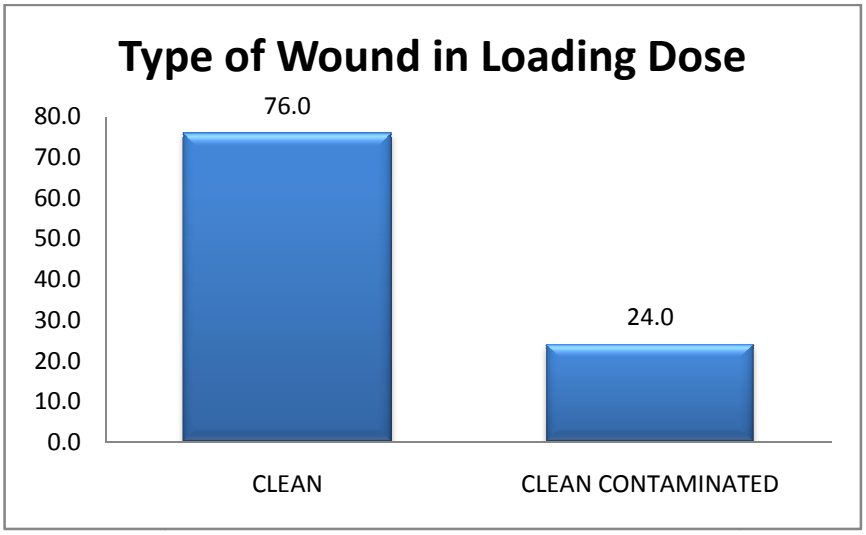


TABLE 19:

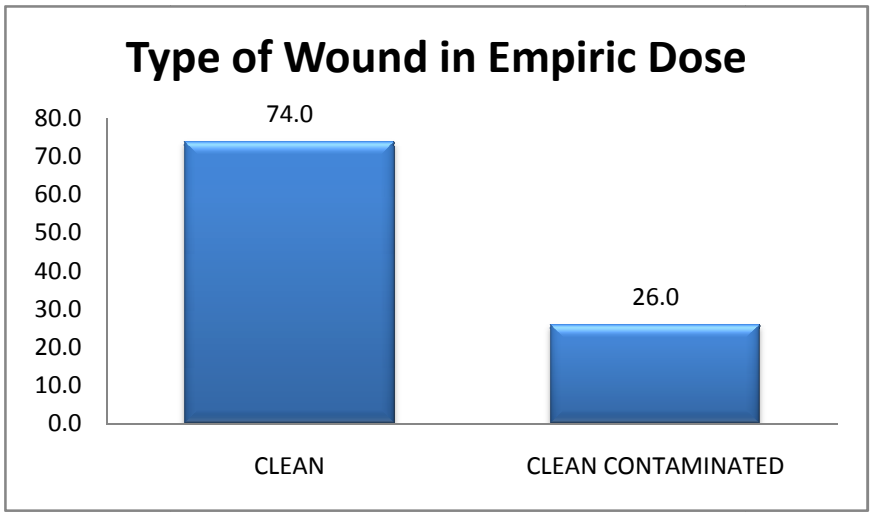
TYPE OF WOUND DISTRIBUTION IN EMPIRIC DOSE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	CLEAN	37	74.0	74.0	74.0
	CLEAN CONTAMINATED	13	26.0	26.0	100.0
	Total	50	100.0	100.0	

Among 50 patients who received empiric dose clean wound were 37[74%] and clean contaminated wound were 13[26%].

GRAPH 16:

TYPE OF WOUND DISTRIBUTION IN EMPIRIC DOSE



DISCUSSION

Perioperative anti-microbial prophylaxis is widely used, and probably overused, for the prevention of SSI. The general principles regarding anti-microbial prophylaxis include. Selection of anti-microbial agents based on the likely pathogens responsible for a SSI with a particular operation

Administration of antibiotics during the induction of anaesthesia such that serum and tissue levels are high at the time of incision and during the course of operation

To achieve high concentrations of antibiotic in the tissues during an operative procedure, the timing of prophylactic antibiotics is critical. A study conducted by

Classen et al[20] showed that subjects who received antibiotics within a two hour period before the incision was made had the lowest incidence of SSI. Several studies conducted by Mangram et al, Bratzler and Hunt, Springer et al and Classen et al[14, 15] showed that use of antibiotics appropriate for the potential pathogen and administration of prophylactic antibiotics within 1 hour before incision reduced the incidence of surgical site infections.

The appropriate duration of antimicrobial therapy for the prevention of postoperative surgical site infection has long been a subject of debate. At present, more than 40 clinical trials have been performed, in which the effectiveness of a single dose of parenteral antimicrobial as a surgical prophylaxis have been studied. Comparison studies of single doses of parenteral antimicrobial versus multiple doses of the same drug, or single dose of antimicrobial agent versus placebo, or single dose of multiple antimicrobials, or a single dose of one agent versus multiple doses of another agent have been carried out. Dipro JT et al[14, 15, 23] in his study proved that the single dose regimens resulted in a similar frequency of postoperative wound infections.

McDonald et al[24] in his study of single versus multiple dose microbial prophylaxis for major surgery, observed that combined odds ratio by both fixed (1.06, 95% CI, 0.89-1.25) and random effects (1.04, 95% CI, 0.86-1.25) models indicated no clear advantage of either single or multiple-dose regimens in preventing SSI.

Scher K[21] conducted a randomized controlled trial in which, patients who were planned to undergoing 801 elective, clean-contaminated operations were assigned to one of the three antibiotic regimens groups:

1) Preoperative 1 g of cefazolin

2) Preoperative 1 g of cefazolin followed by another 1 g dose after 3 hours

3) Preoperative 1 g of cefotetan

Similar surgical site infection rates were noted in all 3 antibiotic regimen groups for those procedures which got completed within 3 hours. The operations which lasted more than 3 hours, resulted in 6.1% infection rate when a single dose of cefazolin was administered and proved to be appreciably greater than 1.3 % infection rates with the two doses of cefazolin or with a single dose of a longer-acting antibiotic, such as cefotetan ($P < 0.01$).

768 patients who were planned to undergo biliary and gastrointestinal tract surgeries were then assigned to one of 2 antibiotic regimens:

1) 1g of cefazolin preoperatively followed by 1g of dose after 3 hours
2) Similar to (1), with 1-g doses every 8 hours for 24 hours

In this study the longer duration of parenteral antibiotic failed to improve the rate of surgical site infection when compared to the use of only perioperative antibiotic coverage. Therefore his study concluded that a single preoperative dose of antibiotic is adequate as operative prophylaxis

when surgery is completed within 3 hours. However for operations of longer duration the period of antibiotic coverage must be extended. Equally effective is a second dose of antibiotic or a single dose of a preoperative antibiotic with extended half-life. Hence there was no value of giving antibiotics after the surgery had been completed.

Mohri Y et al[25] conducted a study in Mie University Graduate School of Medicine, Japan comparing a single dose with a multiple dose regimen of anti-microbial prophylaxis for prevention of surgical site infection between May 2001 and December 2004. It was found that surgical site infection was seen in 9.5 per cent in the first group and in 8.6 per cent in the second group. Thus they concluded that incidence of surgical site infection in elective gastric cancer surgery was similar with both antibiotic prophylaxis regimens.

Fonseca SN et al[26] conducted a study in Brazil from February 2002 to August 2003 by replacing a 24-hour regimen with a single antibiotic prophylaxis for elective surgery. 12299 patients were followed up during their hospital stay. They found that the rate of surgical site infection did not change. Thus they concluded that one-dose antibiotic prophylaxis did not lead to an increase in rates of surgical site infection.

Oostvogel HJ et al[28] conducted a prospective, randomized double-blind trials to investigate the effectiveness of a single dose antibiotic regimen for preventing post-operative wound infection at St Elisabeth Hospital in Netherlands. Patients undergoing "clean-contaminated", "contaminated" or "clean" surgeries were included. Single-dose (pre-operative) prophylaxis was compared with short-term prophylaxis (1 dose pre-operatively and 2 doses post-operatively). They found that the incidence of wound infection was 1.8 % in the short-term group and 3.1% in the single-dose group. The difference was not statistically significant. Thus they concluded that single-dose of antibiotic prophylaxis lowered the rates of post-operative wound infection, even in "clean-contaminated" or "contaminated" cases.

The present study had infection rates of 7.9% and 8.3% in class I and class II respectively among those who received only pre-operative antibiotic prophylaxis. Whereas in those that received post-operative empiric therapy the infection rates were found to be 5.4% and 7.7% in class I and class II respectively.

On comparing the single dose prophylaxis group with that of the group which received multiple post-operative doses of antibiotics, the p value was found to be 0.695 and thus it was concluded that there was no statistical significance between the two groups.

STUDY	PERCENTAGE OF SSI	P VALUE
MOHRI ET AL	EMPIRIC – 8.6% PROPHYLACTIC –9.5%	**<0.05**
OSSTOVOGEL ET AL	PROPHYLACTIC –3.1%	**<0.05**
FONSECA ET AL	FONSECA ET AL 5.4% and 7.7% 5.4% and 7.7%	**<0.05**
PRESENT STUDY	PROPHYLACTIC -7.9% and 8.3%	**<0.05**

CONCLUSION

Our study shows that a loading dose of antibiotics during induction of anaesthesia in clean and clean contaminated surgeries is as effective in preventing post-operative surgical site infection as in the controlled group who received empiric antibiotic of full course in the post operative period for 5-7 days.

The rate of surgical site infections was in no way increased in patients who received a loading dose of antibiotic during induction of anaesthesia in comparison to those who received multiple doses of antibiotics post-operatively. The p value was found to be 0.695 (>0.05), which was not significant.

Thus it can be concluded from this study that a loading dose of antibiotic at the time of induction of anaesthesia is sufficient to prevent post-operative surgical site infections in clean and clean contaminated surgeries thus we can reduced the duration of hospital stay, the cost effectiveness, the adverse effects of antibiotics, nosocomial...

SUMMARY

The study was conducted on 100 patients who underwent either clean or clean contaminated elective general surgical procedures at Coimbatore Medical College Hospital, between june 2013 and june 2014. 50 of whom received a loading dose of antibiotic (injection cefotaxime 2gm IV stat) at the time of induction of anaesthesia and 50 received multiple doses of antibiotics post-operatively. Occurrence of post-operative wound infection was noted among those who received only loading dose of antibiotic at the time of induction of anaesthesia and those who received post-operative empiric therapy.

Statistical analysis was done accordingly, P-value less than 0.05 was considered significant.On analysis there was no statistically significant difference between the loading dose group and empiric group in both clean and clean contaminated surgeries.

BIBLIOGRAPHY

Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection. Infect Control Hosp Epidemiol; 1999. p. 247-78.

Fraenkel G. Some historical notes on infection in surgery. In: Watts MJ, Infection in surgery – basic and clinical aspects. New York; 1981. p. 27-37.

Ananthnarayan R, Panicker CKJ. Sterilisation and disinfection, Textbook of Microbiology. 5th ed. Chennai: Orient Longman Publications; 2000. p. 22-32.

Dunn DL. Diagnosis and treatment of infection. Surgery, basic science and clinical evidence. New York: Springer-Verlag, Inc; 2001. p. 193-216.

Brown RB, Bradley S, Opitz E, Cipriani D, Pieczarka R, Sands M. Surgical wound infections documented after hospital discharge. Am J Infect Control 1987; 15:54 -8.

Schoen F J, Robbins LS, Kumar V, Cotran RS. Wound healing: Robbins' Pathologic Basis of Disease. 5th ed. Philadelphia, PA: WB Saunders Co; 1994. p. 85-91.

Brooks GF, But el JS, Morse SA. Immunology: Jawetz, Melnick and Adelberg's Medical Microbiology. 21st ed. Tata McGraw-Hill Companies; 1999 . p. 110-33.

Zabel DD, Hunt TK, Mueller RV, Goodson III WH. Wound healing. In: Way LW, Doherty GM, Current surgical diagnosis and treatment. Tata McGraw-Hill Companies; 2 003. p. 86-98.

Cobb JP, Schmieg R, Hunt TK, Mundy LM. Inflammation, infection and antibiotics. I n: Way LW, Doherty, Current surgical diagnosis and treatment. Tata McGraw-Hill Companies; 2003. p. 112-41.

Centers for disease control and prevention. National Nosocomial Infection Surveillance (NNIS). Report, data summary from October 1986 to April 1996, issued May 1996. A report from the NNIS System. Am J Infect Control 1996; 24:380-8.

Hai AA, Shrivastava RB. Surgical infections. ASI Textbook of Surgery. Tata McGraw-Hill Co L td; 2003. p. 107-51.

Dellinger EP. Surgical infections and choice of antibiotics. In: Sabiston DC, Textbook of Surgery. The biological basis of modern surgical practice. 16th ed. Philadelphia: WB Saunders Co; 2001. p. 171-88.

Cruse PJ. Surgical wound infection. In: Wonsiewicz MJ, Infections diseases. Philadelphia: W B Saunders Co; 1992. p. 758-64.

Patel NP, Malangoni MA. Antimicrobial agents for surgical infections. Surg Clin N Am 2009; 89: 327-47.

Kirby JP, Mazuski JE. Prevention of surgical site infection. Surg Clin N Am 2009; 89: 365-90

Anonymous. ASHP Therapeutic guidelines on antimicrobial prophylaxis in surgery. Am J Health Syst Pharm 1999;56: 1839-88

Platt R, Zaleznik DF, Hopkins CC, et al. peri-operative antibiotic prophylaxis for herniorrhaphy and breast surgery. N Engl J Med 1990;322: 153-60

Nichols RL, Condon RE, Barie PS. Antibiotic prophylaxis in surgery-2005 and beyond. Surg Infect (Larchmt) 2005;6;349-61

Burke JF. The effective period of preventive antibiotic action in experimental incisions and dermal lesions. Surgery 1961;50:161-8

Classen DC, Evans RS, Pestotnik SL, et al. The timing of prophylactic administration of antibiotics and the risk of surgical wound infection. N Engl J Med 1992;326:281-6

Scher K. Studies on the duration of antibiotic administration for surgical prophylaxis. Am Surg 1997;63:59-62

Barie PS. Modern surgical antibiotic prophylaxis and therapy-less is more. Surg Infect (Larchmt) 2000;1:23-9

DiPiro JT, Cheung RP, Bowden TA Jr, et al. Single dose systemic antibiotic prophylaxis of surgical wound infections. Am J Surg 1986;152:552-9

McDonald M, Grabsch E, Marshall C, et al. Single versus multiple dose antimicrobial prophylaxis for major surgery: a systematic review. Aust N Z J Surg 1998;68:388-96

Mohri Y, Tonouchi H, Kobayashi M, Nakai K, Kusunoki M. Randomised clinical trial of single-dose versus multiple dose anti-microbial prophylaxis in gastric cancer surgery. Br J Surg 2007; 94(6):683-8

Fonseca SN, Kunzle SR, Junqueira MJ, Nascimento RT, de Andrade JI, Leven AS. Implementing 1-dose antibiotic prophylaxis for prevention of surgical site infection. Arch Surg 2006; 141(11):1109-14

Scher KS, Wroczynski AF, Jones CW. Duration of antibiotic prophylaxis. An experimental study. Am J Surg 1986; 151(2):209-12

Oostvogel HJ, van Vroonhoven TJ, van der Werken C, Lenderink AW. Single-dose versus short-term antibiotic therapy for prevention of wound infection in general surgery. A prospective, randomized double-blind trial. Acta Chir Scand1987; 153(10):571-5

Yousuf M, Hussain M. Need and duration of antibiotic therapy in clean and clean contaminated operations. J Pak Med Assoc 2002; 52(7):284-7

Ali M, Raza A. Role of single dose antibiotic prophylaxis in clean orthopedic surgery. J Coll Physicians Surg Pak 2006; 16(1):45-8

Zahid M A, Bakhsh R, Dar FS, Akhter N, Malik ZI. Comparison of single dose and three dose antibiotic prophylaxis with cefotaxime sodium in cholecystectomy. J Ayub Med Coll Abbottabad 2003; 15(1):38-40

Yang Z. Short-term versus long-term anti-microbial prophylaxis in abdominal surgery: a multi-center open randomized comparative trial. Zhonghua Wai Ke Zha Zhi 2001; 39(10):770-2

Shea, Apic, CD C, SIS. Consensus paper on the surveillance of surgical wound infection. Infect Control Hosp Epidemiol 1992; 13(10):599-605.

Gil-Egea MJ, Pi-Sunyer MT, Verdaguer A, Sanz F, Sitges-Serra A, Eleizegui LT. Surgical wound infections: prospective study of 4,486 clean wounds. Infect Control 1987;8(7):277-80.

Haley RW, Culver DH, Morgan WM, White JW, Emori TG, Hooton TM. Identifying patients at high risk of surgical wound infections. A simple multivariate index of patient susceptibility and wound contamination. Am J Epidemiol 1995;121:206-15.

Garibaldi RA, Cushing D, Lerer T. Risk factors for postoperative infection. Am J Med 1991;91(Suppl 3B):1585-635.

Morro ML, Carrieri MP, Tozzi AE, Lona S, Greco D. Risk factors for surgical wound infections in clean surgery: a multicenter study. Italian Prinos Study Group Ann Ital Chir 1996;67:13-9.

Anonymous. Antimicrobial prophylaxis in surgery. Med Lett Drugs Ther 1997;39(1012):97-102.

Nichols RL. Antibiotic prophylaxis in surgery. J Chemother 1989;1(3):170-8.

Nichols RL. Surgical antibiotic prophylaxis. Med Clin North Am 1995;79(3):509-22.

Committee on control of surgical infections of the Committee on pre and postoperative care. American College of Surgeons. Manual on Control of Infection in Surgical Patients. Philadelphia: JB Lippincott Co; 1984.

Condon RE, Schulte WJ, Malangoni MA, Anderson Teschendorf MJ. Effectiveness of a surgical wound surveillance program. Arch Surg 1983;118:303-7.

QCulver DH, Horan TC, Goynes RP, Martone WJ, Jarvis WR, Emori TG, et al. Surgical wound infection rates by wound class, operative procedure and patient risk index. NNIS System. Am J Med 1991;(Suppl 3B):152S-7S.

Gupta R, Sinnette D, Carpenter R, Preece PE, Royle GT. Antibiotic Prophylaxis for postoperative wound infection in clean elective breast surgery. Eur J Surg Oncol 2000; 26 : 363-6.

Philip S, Barie, et al. Surgical site infections. Surg Clin N Am 2005; 85:1115-1135.

ANEXURES

SAMPLE INFORMED CONSENT FROM COIMBATORE MEDICAL COLLEGE HOSPITAL ,COIMBATORE.

TITLE OF THE PROJECT - COMPARATIVE STUDY OF LOADING DOSE OF ANTIBIOTIC AT THE TIME OF INDUCTION OF ANAESTHESIA VERSUS EMPIRIC ANTIBIOTICS IN CLEAN AND CLEAN-CONTAMINATED ELECTIVE GENERAL SURGICAL CASES

PRINCIPAL INVESTIGATOR - DR.R.RANGANATHAN

GUIDE - DR.S.SARADHA. M.S.,

PROFESSOR OF SURGERY

PURPOSE OF RESEARCH

I have been informed that this study is a comparison of loading dose of antibiotic at the time of induction of anaesthesia versus empiric antibiotics in clean and clean contaminated general surgerycases. I have also been given a free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

PROFORMA

SCHEME OF CASE TAKING:

1)	Name:	CASE NO:
2)	Age:	IP NO:
3)	Sex:	DOA:
4)	Religion:	DOS:
5)	Occupation:	DOD:

Residence:

Chief complaints

Provisional diagnosis

Past History:

Diabetes mellitus

Hypertension

History of any drug intake

General Physical Examination

Pallor	present/absent
Icterus	present/absent
Clubbing	present/absent
Generalized Lymphadenopathy	present/absent
Build	Poor/Middle /Well
Nourishment	Poor / Middle / Well

11) Vitals

PR:

BP:

RR:

Temp:

Weight:

Other Systemic Examination:

Respiratory System

Cardiovascular System

Central Nervous System

Investigation:

Blood: Hb

TC

DC

ESR

BT

CT

BLOOD UREA

SERUM CREATININE

RBS

Urine:

Albumin

Sugar

Micro

Final Diagnosis:

Details of Surgery:

Diagnosis	Operative Procedure	Duration of Procedure

16) Follow up:

Local examination of wound

	POD2	POD5	POD8
Clean			
Seroma			
Edema			
Erythema			
dema			
Erythema			
Tenderness			
Abscess			
Pus discharge			
Gaping of wound			

Pus culture and sensitivity: positive negative

Resistance to the antibiotic used: yes no

17) Inference:

18) Comments:

MASTER CHART

GROUP A-SINGLE DOSE							LOADING DOSE	
S.NO	NAME	AGE/SEX	IPNO	DIAGNOSIS	TYPE OF SURGERY	Type of wound	SSI	TYPE OF SSI
1	SUBRAMANIAM	50/M	52232	[R]DIRECT INGUINAL HERNIA	[R] HERNIOPLASTY	CLEAN	NIL	
2	JAGAJOTHY	43/F	51164	FIBROADENOMA [L]BREAST	EXCISION	CLEAN	NIL	
3	PALANATHAL	38/F	48460	FIBROADENOMA [L]BREAST	EXCISION	CLEAN	NIL	
4	VIGNESH	14/M	50685	ATROPHIC [R]TESTIS	[R]ORCHIDECTOMY	CLEAN	NIL	
5	SAROJA	33/F	53382	[LT]FIBROADENOMA BREAST	EXCISION	CLEAN	NIL	
6	PARAMASIVAM	60/M	32874	[L]INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
7	SIVASAMY	64/M	51554	B/L DIRECT INGUINAL HERNIA	B/LHERNIOPLASTY	CLEAN	PRESENT	PURULENT
8	KATHIRESAN	75/M	45576	B/L DIRECT INGUINAL HERNIA	B/LHERNIOPLASTY	CLEAN	NIL	
9	NELSON	65/M	43956	[L] GYNAECOMASTIA	[L]WEBSTERS PROCEDURE	CLEAN	NIL	
10	MEENATCHI	51/M	43961	[R]INDIRECT INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
11	MANOJ	31/M	42362	[L] INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
12	KUMAR	38/M	38934	B/L VARICOSE VEINS	TRENDELENBERG PROCEDURE	CLEAN	NIL	
13	MOHAN	34/M	40657	[L]INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
14	MOHAMMED BILLAL	15/M	37433	[L] GYNAECOMASTIA	WEBSTERS PROCEDURE	CLEAN	NIL	
15	SENTHIL KUMAR	43/F	37356	[R] INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	PRESENT	SEROUS
16	MOTHIRAM	35/M	32495	THYROGLOSSAL CYST	SISTRUNK PROCEDURE	CLEAN	NIL	
17	ANGURAJ	44/M	27901	[R] INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
18	ARUCHAMY	65/M	24669	B/L DIRECT INGUINAL HERNIA	B/LHERNIOPLASTY	CLEAN	NIL	
19	RAJENDRAN	45/M	24656	[L]DIRECT INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
20	RATHINAMOORTHY	55/M	23527	[R]DIRECT INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
21	MARIMUTHU	18/M	23283	[R]GYNAECOMASTIA	[R]WEBSTERS PROCEDURE	CLEAN	NIL	
22	SATHISH KUMAR	22/M	23313	[R]INDIRECT INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	PRESENT	WOUND GAPING
23	MURUGAN	65/M	20211	[L]HYDROCELE	[L]JABOLEYS REPAIR	CLEAN	NIL	

24	PRABHU	27/M	20112	[R]DIRECT INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
25	THANGARAJ	35/M	18698	[L]INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
26	JOHAN	65/M	18629	[L]INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
27	AVINASHILINGAM	65/M	30043	B/LINGUINAL HERNIA	B/LHERNIOPLASTY	CLEAN	NIL	
28	BALASUBRAMANI	62/M	31064	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
29	MUNIANDI	35/M	31112	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
30	BADRASAMI	26/M	30319	B/LGYNAECOMASTIA	B/LWEBSTERS PROCEDURE	CLEAN	NIL	
31	SALEEM	35/M	29848	[L]INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
32	JUSTIN	42/M	28712	B/LINGUINAL HERNIA	B/LHERNIOPLASTY	CLEAN	NIL	
33	LURDASAMY	66/M	28097	[L]INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
34	KALAIARASI	27/F	47051	UMBILICAL HERNIA	MESH REPAIR	CLEAN	NIL	
35	SHANTHAMANI	45/F	47231	MNG THYROID	SUBTOTAL THYROIDECTOMY	CLEAN	NIL	
36	RUBA	26/F	45582	[L]FIBROADENOMA	EXCISION	CLEAN	NIL	
37	SHAJ	16/F	37394	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
38	SUSEELA	42/F	41736	[L]INGUINAL HERNIA	[L]HERNIOPLASY	CLEAN	NIL	
39	THILAGA	88/F	48998	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
40	BHUVANESHWARI	28/F	47033	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	PRESENT	ERYTHEMA
41	JOTHIMANI	45/F	21755	CALCULUS CHOLECYSTITIS	OPEN CHOLECTSTECTOMY	CLEAN CONTAMINATED	NIL	
42	GUNASEKARAN	18/M	37396	CALCULUS CHOLECYSTITIS	LAP CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
43	AJITHKUMAR	15/M	30621	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
44	ABBAS	20/M	24690	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
45	RAMESH	33/M	52115	SUBACUTE INTESTINAL OBSTRUCTION	DIAGNOSTIC LAPROSCOPY	CLEAN CONTAMINATED	NIL	

46	SUDHA	36/F	44589	GALL BLADDER POLYP	LAP CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
47	LAKSHMI	60/F	37613	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
48	SAIYATH	13/M	42664	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
49	KANDASAMY	45/M	39128	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
50	ASAI THAMBI	26/M	26901	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	

GROUP B-EMPIRIC DOSE								
1	RAMASAMY	49/M	42606	B/L DIRECT INGUINAL HERNIA	B/L HERNIOPLASY	CLEAN	NIL	
2	KUMAR	27/M	42792	[R]INGUINAL HHERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
3	SHANMUGAM	62/M	38504	[L]INGUINAL HERNIA	[L]HERNIOPLASTY	CLEAN	NIL	
4	GEETHANJALI	30/F	47832	FIBROADENOMA[R]BREAST	EXCISION	CLEAN	NIL	
5	KITTAN	45/M	26557	[L]INGUINAL HERNIA	[L]HERNIOPLASTY	CLEAN	NIL	
6	ARPUTHARAJ	62/M	22395	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
7	GOWRI	45/M	44194	CA[R]BREAST	[R]MRM	CLEAN	NIL	
8	SAVITHA	48/F	36271	SOLITARY NODULE THYROID	[R]HEMITHYROIDECTOMY	CLEAN	NIL	
9	SARAVANAN	44/M	46852	B/L DIRECT INGUINAL HERNIA	B/L HERNIOPLASY	CLEAN	NIL	
10	BIPONTHABA	26/M	47091	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
11	MOORTHY	50/M	45345	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
12	CHANDRASEKAR	46/M	43792	[L]INGUINAL HERNIA	[L]HERNIOPLASTY	CLEAN	NIL	
13	AMSAVENI	20/F	51351	FIBROADENOMA[R]BREAST	EXCISION	CLEAN	PRESENT	SREOUS
14	KOPPAMMAL	50/F	48298	[R]SOLITARY NODULE THYROID	[R]HEMITHYROIDECTOMY	CLEAN	NIL	
15	RAJALAKSHMI	35/F	35851	TOXIC NODULE THYROID	NEAR TOTAL THYROIDECTOMY	CLEAN	NIL	
16	KANNAKI	52/F	37136	[R]CA BREAST	[R]MRM	CLEAN	NIL	
17	JESI	36/F	49560	[L]HEMITHYROIDECTOMY		CLEAN	NIL	
18	GOKULAMMAL	35/F	41984	[R]CA BREAST	[R]MRM	CLEAN	NIL	
19	MHIRAISA	45/F	36911	[L]FOLLICULAR ADENOMA	[L]HEMITHYROIDECTOMY	CLEAN	NIL	
20	SAMPATH KUMAR	45/M	52900	[L]VARICOSE VEIN	[L]TRENDELENBERG PROCEDURE	CLEAN	NIL	
21	GANESH RAJA	40/M	43076	[L]VARICOSE VEIN	[L]TRENDELENBERG PROCEDURE	CLEAN	NIL	
22	MAHESHWARI	28/F	46134	[R]VARICOSE VEIN	[R]TRENDELENBERG PROCEDURE	CLEAN	NIL	
23	MUTHUMARI	52/F	42966	CA[R]BREAST	[R]MRM	CLEAN	NIL	
24	RATHINASELVI	21/F	33171	FIBROADENOMA[R]BREAST	EXCISION	CLEAN	NIL	
25	SELVARAJ	44/M	28557	UMBLICAL HERNIA	MESH REPAIR	CLEAN	NIL	

26	AUGUSTIN	16/M	49299	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
27	KARUNAIYAPPAN	66/M	19228	[R]HYDROCELE	[R]JABOLEYS PROCEDURE	CLEAN	NIL	
28	KANNADASAN	35/M	17757	UMBLICAL HERNIA	MESH REPAIR	CLEAN	NIL	
29	PALANIAMMAL	65/F	37654	CA[R]BREAST	[R]MRM	CLEAN	NIL	
30	JENITHA	27/F	32514	FIBROADENOMA[L]BREAST	EXCISION	CLEAN	NIL	
31	KALAMANI	47/F	38256	MNG THYROID	SUBTOTAL THYROIDECTOMY	CLEAN	NIL	
32	NIRMALA	50/F	22553	CA[L]BREAST	[L]MRM	CLEAN	NIL	
33	JAYA	35/F	20704	SOLITARY NODULE THYROID	[R]HEMITHYROIDECTOMY	CLEAN	NIL	
34	SHAJAHAN	44/M	49278	[R]INGUINAL HERNIA	[R]HERNIOPLASTY	CLEAN	NIL	
35	RAMASAMY	75/M	49433	[L]INGUINAL HERNIA	[L]HERNIOPLASTY	CLEAN	NIL	
36	DURAISAMY	58/M	44947	B/L DIRECT INGUINAL HERNIA	B/L HERNIOPLASY	CLEAN	PRESENT	ERYTHEMA
37	GUNASEKARAN	56/m	43260	B/L INGUINAL HERNIA	B/L HERNIOPLASY	CLEAN	NIL	
38	PUSHPA	40/F	44840	CALCULUS CHOLECYSTITIS	LAP CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
39	GOMATHI	27/F	46151	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
40	PRIYA	18/F	44599	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
41	SUGUNA	15/F	41357	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
42	PALANISAMY	69/M	46268	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
43	SUBRAMANI	48/M	44603	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
44	REKHA	27/F	30497	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
45	PALANISAMY	29/M	45979	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	PRESENT	ERYTHEMA
46	MOHANRAJ	60/M	43218	CHOLELITHIASIS	OPEN CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
47	SUNDARASAMY	58/M	41561	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
48	KALIAMMAL	45/F	46611	CHOLELITHIASIS	OPEN CHOLECYSTECTOMY	CLEAN CONTAMINATED	NIL	
49	PADMAVATHY	15/F	45090	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	
50	MUNIAPERUMAL	33/M	52707	SUBACUTE APPENDICITIS	LAP APPENDICECTOMY	CLEAN CONTAMINATED	NIL	

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது அறுவை சிகிச்சை துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவன் அவர்கள் மேற்கொள்ளும் **"தேர்ந்தெடுத்த சுத்தமான மற்றும் சுத்தம் சற்றே குன்றிய அறுவை சிகிச்சையின் பொழுது மயக்க மருந்து கொடுக்கும் முன் மொத்தமாக நுண்ணுயிர் கொல்லியை செலுத்துதல் மற்றும் வழக்கத்தில் உள்ள நுண்ணுயிர் கொல்லி வரைமுறைகளை ஒப்பிட்டு நோக்கும் ஆய்வு"** குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடன், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்தில் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :